

```

=> file biosis caba caplus embase japiro lifesci medline scisearch
=> e lubitz werner/au
E1      3      LUBITZ W D/AU
E2      1      LUBITZ W J/AU
E3      393 --> LUBITZ WERNER/AU
E4      1      LUBITZ WERNER PROF/AU
E5      1      LUBITZ WILLIAM/AU
E6      2      LUBITZ WILLIAM DAVID/AU
E7      1      LUBITZ WOLFGANG/AU
E8      420     LUBITZ WOLFGANG/AU
E9      1      LUBITZKI LOTHAR/AU
E10     1      LUBITZOMERO C/AU
E11     7      LUBITZSCH PETER/AU
E12     1      LUBITZSCH WOLFGANG/AU

=> s el-e4 and bacter? and ghost?
L1      151 ("LUBITZ W D"/AU OR "LUBITZ W J"/AU OR "LUBITZ WERNER"/AU OR
          "LUBITZ WERNER PROF"/AU) AND BACTER? AND GHOST?

=> dup rem 11
PROCESSING COMPLETED FOR L1
L2      57 DUP REM L1 (94 DUPLICATES REMOVED)
=> s bioaffinity and (binding pair)
L4      1 BIOAFFINITY AND (BINDING PAIR)

=> d

L4      ANSWER 1 OF 1 CAPLUS COPYRIGHT 2009 ACS on STN
AN      1990:512008 CAPLUS <>LOGINID::20091202>>
DN      113:112008
OREF 113:18897a,18900a
TI      Application of poly(ethyleneimine) derivatized with a hydrophobic group in
protein immobilization for immunoassays and ***bioaffinity***  

separations
IN      Lau, Philip Hon Peng
PA      du Pont de Nemours, E. I., and Co., USA
SO      Eur. Pat. Appl., 12 pp.
CODEN: EPXXDW
DT      Patent
LA      English
FAN.CNT 1
    PATENT NO.           KIND    DATE      APPLICATION NO.      DATE
    -----  -----
PI      EP 341498           A1    19891115    EP 1989-107607    19890427
      EP 341498           B1    19940518
      R: DE, FR, GB, IT
      US 4952519          A    19900828    US 1988-188956    19880502
      JP 02043947         A    19900214    JP 1989-112314    19890502
      JP 07034859         B    19950419
PRAI US 1988-188956       A    19880502
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OSC.G  5      THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)

=> s l2 and (biotin or streptavidin or avidin or hapten or saccharide or lectin
or ligand or receptor)
L5      3 L2 AND (BIOTIN OR STEPTAVIDIN OR AVIDIN OR HAPten OR SACCHARIDE

```

OR LECTIN OR LIGAND OR RECEPTOR)

> d 1-

YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):Y

L5 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 2005:120755 CAPLUS <>LOGINID::20091202>>
 DN 142:225686

TI Sealing of ***bacterial*** ***ghosts*** for drug delivery using
 membrane vesicles and affinity ***ligand*** interactions

IN ***Lubitz, Werner***

PA Austria

SO PCT Int. Appl., 37 pp.

CODEN: PIIXD2

DT Patent

LA German

FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------|--|------|----------|------------------|----------|
| PI | WO 2005011713 | A1 | 20050210 | WO 2004-EP8790 | 20040805 |
| | W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| | DE 10335796 | A1 | 20050303 | DE 2003-10335796 | 20030805 |
| | AU 2004260620 | A1 | 20050210 | AU 2004-260620 | 20040805 |
| | AU 2004260620 | B2 | 20080124 | | |
| | CA 2534612 | A1 | 20050210 | CA 2004-2534612 | 20040805 |
| | EP 1656149 | A1 | 20060517 | EP 2004-763831 | 20040805 |
| | R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK | | | | |
| | NZ 545232 | A | 20081224 | NZ 2004-545232 | 20040805 |
| | US 20060286126 | A1 | 20061221 | US 2006-567426 | 20060516 |
| PRAI | DE 2003-10335796 | A | 20030805 | | |
| | WO 2004-EP8790 | W | 20040805 | | |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2000:623585 CAPLUS <>LOGINID::20091202>>

DN 133:227782

TI ***Bacterial*** ***ghosts*** as carrier and targeting vehicles

IN Huter, Veronika; ***Lubitz, Werner***

PA Austria

SO Ger. Offen., 10 pp.

CODEN: GWXXBX

DT Patent

LA German

FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|-------|---|--|----------------------------------|---|----------------------------------|
| PI | DE 19909770 CA 2370714 WO 2000053163 | A1 A1 A1 | 20000907 20000914 20000914 | DE 1999-19909770 CA 2000-2370714 WO 2000-EP1906 | 19990305 20000303 20000303 |
| | W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG | | | | |
| EP | 1158966 | A1 | 20011205 | EP 2000-912549 | 20000303 |
| EP | 1158966 | B1 | 20030611 | | |
| | R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO | | | | |
| JP | 2002538198 | T | 20021112 | JP 2000-603652 | 20000303 |
| AT | 242630 | T | 20030615 | AT 2000-912549 | 20000303 |
| NZ | 514408 | A | 20040130 | NZ 2000-514408 | 20000303 |
| AU | 778166 | B2 | 20041118 | AU 2000-34272 | 20000303 |
| PRAI | DE 1999-19909770 WO 2000-EP1906 | A W | 19990305 20000303 | | |
| OSC.G | 4 | THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS) | | | |

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|-------|---|--|----------------------------------|-----------------|----------|
| PI | WO 9113155 | A1 | 19910905 | WO 1991-EP308 | 19910219 |
| | W: AU, FI, HU, JP, SU, US RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE | | | | |
| DE | 4005874 | A1 | 19911107 | DE 1990-4005874 | 19900224 |
| AU | 9172373 | A | 19910918 | AU 1991-72373 | 19910219 |
| EP | 516655 | A1 | 19921209 | EP 1991-903789 | 19910219 |
| EP | 516655 | B1 | 19940504 | | |
| | R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE | | | | |
| JP | 05503014 | T | 19930527 | JP 1991-503980 | 19910219 |
| JP | 3238396 | B2 | 20011210 | | |
| AT | 105335 | T | 19940515 | AT 1991-903789 | 19910219 |
| US | 5470573 | A | 19951128 | US 1992-924028 | 19920930 |
| PRAI | DE 1990-4005874 EP 1991-903789 WO 1991-EP308 | A A A | 19900224 19910219 19910219 | | |
| OSC.G | 8 | THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS) | | | |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

OSC.G 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s bacter? and ghost? and (biotin or streptavidin or avidin or hapten or saccharide or lectin or ligand or receptor)
L6 107 BACTER? AND GHOST? AND (BIOTIN OR STEPTAVIDIN OR AVIDIN OR HAPTE N OR SACCHARIDE OR LECTIN OR LIGAND OR RECEPTOR)

=> dup rem 16
PROCESSING COMPLETED FOR L6
L7 68 DUP REM L6 (39 DUPLICATES REMOVED)

=> d bib ab kwic 1-
YOU HAVE REQUESTED DATA FROM 68 ANSWERS - CONTINUE? Y/(N):y

L7 ANSWER 1 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 2009:589834 BIOSIS <>LOGINID:20091202>>
DN PREV200900590937
TI Pharmaceutical Biotechnology.
AU Guzman, CA [Editor]; Feuerstein, GZ [Editor]
SO Guzman, CA [Editor]; Feuerstein, GZ [Editor]. Adv. Exp. Med. Biol., (2009)
Pharmaceutical Biotechnology.
Publisher: SPRINGER-VERLAG BERLIN, HEIDELBERGER PLATZ 3, D-14197 BERLIN,
GERMANY. Series: Advances in Experimental Medicine and Biology.
CODEN: AEMBAP. ISSN: 0065-2598. ISBN: 978-1-4419-1131-5(H).
DT Book
LA English
ED Entered STN: 21 Oct 2009
Last Updated on STN: 21 Oct 2009
AB This 254-page book presents and describes Pharmaceutical Biotechnology. The book is organized into 15 individually authored chapters and these are further divided into different sections. The first chapter deals with translational medicine, a paradigm shift in modern drug discovery and development, the role of biomarkers. The second chapter deals with natural products in drug discovery, present status and perspectives. The third chapter deals with protein pharmaceuticals, discovery and preclinical development. Remaining chapters include the role of nanobiotechnology in drug discovery, conotoxin venom peptide therapeutics, shark novel antigen receptors, immune interventions of human diseases through toll-like receptors, genome-based vaccine development, virus-like particles as a vaccine delivery system and applications of ***bacterial*** ***ghosts*** in biomedicine. The book highlights a list of contributors and their respective institutions. Each chapter contains a list of references. The text is written in English. Users of this book will include biotechnologists, molecular biologists, and pharmacologists.
AB . . . interventions of human diseases through toll-like receptors, genome-based vaccine development, virus-like particles as a vaccine delivery system and applications of ***bacterial*** ***ghosts*** in biomedicine. The book highlights a list of contributors and their respective institutions. Each chapter contains a list of references. . .
IT . . .
system disease, viral disease
Influenza (MeSH)

IT Chemicals & Biochemicals
 cytokine; calcium channel; conotoxin; sodium channel; protein
 pharmaceuticals; immune modulators; toll-like ***receptor*** :
 signaling

L7 ANSWER 2 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 2008:316903 CAPLUS <>LOGINID::20091202>>
 DN 148:306434
 TI Use of glycolipids as adjuvants
 IN Ebensen, Thomas; Morr, Michael; Guzman, Carlos A.; Milkereit, Goetz
 PA Helmholz-Zentrum Fuer Infektionsforschung GmbH, Germany
 SO PCT Int. Appl., 75 pp.
 CODEN: PIXXD2
 DT Patent
 LA English
 FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------|---|------|----------|-----------------|----------|
| PI | WO 2008028667 | A2 | 20080313 | WO 2007-EP7794 | 20070906 |
| | WO 2008028667 | A3 | 20080703 | | |
| | W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA | | | | |
| | EP 1897557 | A1 | 20080312 | EP 2006-18723 | 20060907 |
| | R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU | | | | |
| | AU 2007294103 | A1 | 20080313 | AU 2007-294103 | 20070906 |
| | CA 2661280 | A1 | 20080313 | CA 2007-2661280 | 20070906 |
| | EP 2059257 | A2 | 20090520 | EP 2007-802189 | 20070906 |
| | R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, RS | | | | |
| | IN 2009MN00464 | A | 20090515 | IN 2009-MN464 | 20090305 |
| PRAI | EP 2006-18723 | A | 20060907 | | |
| | WO 2007-EP7794 | W | 20070906 | | |
| OS | MARPAT 148:306434 | | | | |
| AB | The present invention relates to adjuvants of the glycolipid type and their uses in pharmaceutical compns., like in vaccines. In particular, the present invention provides new uses of compds. useful as adjuvants for prophylactic and/or therapeutic vaccination in the treatment of infectious diseases, inflammatory diseases, autoimmune diseases, tumors and allergies. The compds. are particularly useful not only as systemic, but preferably as mucosal adjuvants. | | | | |
| IT | Glycoproteins | | | | |
| | RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses) (CD40-L (antigen CD40 ***ligand***); vaccines comprising antigen and glycolipids as adjuvants induce IgG, IgA, and T-cell responses) | | | | |

IT Eubacteria
 (***ghost*** ***bacteria*** , antigen delivery system; vaccines comprising antigen and glycolipids as adjuvants induce IgG, IgA, and T-cell responses)

L7 ANSWER 3 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 2008:1185246 CAPLUS <>LOGINID::20091202>>
 DN 149:400314

TI Adjuvant combinations comprising a microbial TLR agonist, a CD40 or 4-1BB agonist, and optionally an antigen and the use thereof for inducing a synergistic enhancement in cellular immunity
 IN Delucia, Dave
 PA Regents of the University of Colorado, USA
 SO U.S. Pat. Appl. Publ., 10pp.
 CODEN: USXXCO

DT Patent
 LA English
 FAN.CNT 1

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|----------------------|------|----------|-----------------|----------|
| PI US 20080241139 | A1 | 20081002 | US 2007-931237 | 20071031 |
| PRAI US 2006-863695P | P | 20061031 | | |

AB Adjuvant combinations comprising at least one microbial TLR agonist such as a whole virus, ***bacterium*** or yeast or portion thereof such a membrane, spheroplast, cytoplasm, or ***ghost*** , a CD40 or 4-1BB agonist and optionally an antigen wherein all 3 moieties may be sep. or comprise the same recombinant microorganism or virus are disclosed. The CD40 or 4-1BB agonists preferably comprise an agonistic anti-CD40 antibody or anti-4-1BB antibody. The use of these immune adjuvants for treatment of various chronic diseases such as cancer, allergy, inflammation, infection, and autoimmune disease is also provided.

AB Adjuvant combinations comprising at least one microbial TLR agonist such as a whole virus, ***bacterium*** or yeast or portion thereof such a membrane, spheroplast, cytoplasm, or ***ghost*** , a CD40 or 4-1BB agonist and optionally an antigen wherein all 3 moieties may be sep. or comprise the same. . .

IT Glycoproteins
 RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (CD40-L (antigen CD40 **ligand**); adjuvant combinations comprising a microbial TLR agonist, a CD40 or 4-1BB agonist, and antigen and use thereof for inducing a synergistic enhancement in cellular immunity)

L7 ANSWER 4 OF 68 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
 AN 2008482706 EMBASE <>LOGINID::20091202>>

TI Preventing recurrent urinary tract infections: Role of vaccines.
 AU Zakri, Rhana Hassan; DasGupta, Ranan; Dasgupta, Prokar; Khan, Mohammad Shamim
 CS Department of Urology, Guy's and St. Thomas' NHS Foundation Trust, King's London School of Medicine, London, United Kingdom. rhzakri@doctors.org.uk
 AU Zakri, Rhana Hassan
 CS Urology, Guy's and St. Thomas' NHS Foundation Trust, Great Maze Pond, London SE1 9RJ, United Kingdom. rhzakri@doctors.org.uk
 AU Zakri, R. H. (correspondence)
 CS Urology, Guy's and St. Thomas' NHS Foundation Trust, Great Maze Pond, London SE1 9RJ, United Kingdom. rhzakri@doctors.org.uk

SO BJU International, (November 2008) Vol. 102, No. 9, pp. 1055-1056.
Refs: 12
ISSN: 1464-4096; E-ISSN: 1464-410X CODEN: BJINFO

PB Blackwell Publishing Ltd, 9600 Garsington Road, Oxford, OX4 2XG, United Kingdom.

CY United Kingdom

DT Journal; Note

FS 004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
017 Public Health, Social Medicine and Epidemiology
028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles

LA English

ED Entered STN: 4 Nov 2008
Last Updated on STN: 4 Nov 2008

CT Medical Descriptors:
 bacterial strain
 clinical trial
 Escherichia coli
 follow up
 human
 hysterectomy
 nonhuman
 note
 priority journal
 prophylaxis
 pyelonephritis: DT, drug therapy
 pyelonephritis: PC, prevention
 recurrent disease
 spinal cord injury
 unspecified side effect: SI, side effect
 upregulation
 *urinary tract infection: DT, drug therapy
 *urinary tract infection: PC, prevention
 vaccination
 vagina mucosa
 bacterial protein: EC, endogenous compound
 ****bacterial vaccine: DT, drug therapy***
 ****bacterial vaccine: NA, intranasal drug administration***
 ****bacterial vaccine: PD, pharmacology***
 ****bacterial vaccine: SC, subcutaneous drug administration***
 immunoglobulin G: EC, endogenous compound
 outer membrane protein A: EC, endogenous compound
 *papdg vaccine: DT, drug therapy
 placebo
 protein fepa: EC, endogenous compound
 ****siderophore receptor iron: PD, pharmacology***
 ****siderophore receptor iron: SC, subcutaneous drug administration***
 *solco urovac: AE, adverse drug reaction
 *solco urovac: CT, clinical trial
 *solco urovac: DT, drug therapy
 *solco urovac: PA, parenteral drug administration
 unclassified drug
 ****vibrio cholerae ghosts vaccine: NA, intranasal drug***
*** administration***

AN 2007:1469963 CAPLUS <<LOGINID::20091202>>
 DN 148:99092
 TI Immunogenic multivalent adhesins preparation and use as vaccines
 IN Knight, Stefan
 PA Swed.
 SO PCT Int. Appl., 40pp.
 CODEN: PIIXXD2
 DT Patent
 LA English
 FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------|---|------|----------|-----------------|----------|
| PI | WO 2007148229 | A2 | 20071227 | WO 2007-IB2430 | 20070222 |
| | WO 2007148229 | A3 | 20081030 | | |
| | W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BE, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA | | | | |
| PRAI | US 2006-775678P | P | 20060222 | | |

AB Immunogenic multivalent complexes comprise a ***receptor*** -binding domain of a two-domain adhesin antigen or a single chain polyadhesin antigen coupled with a carrier particle. The antigen and the carrier particle are coupled by an affinity-tag system that uses a small peptide tag and that allows a 1-step affinity purifn. The immunogenic multivalent complex further comprises a flexible linker between the C terminus of the antigen and the coupling tag. Immunostimulating and adhesion-blocking agents, vaccines, immunogenic formulations, and immunogenic constructs and compns. comprise an immunogenic multivalent complex. Methods for identifying a two-domain adhesin antigen comprise selecting a sequence of a pilin from a chaperone/usher system; searching a protein and/or DNA sequence database with the pilin sequence; and identifying a sequence that aligns to the C-terminal portion of the pilin sequence and comprises an unmatched sequence of from about 140 to about 240 amino acid residues preceding the aligned region.

AB Immunogenic multivalent complexes comprise a ***receptor*** -binding domain of a two-domain adhesin antigen or a single chain polyadhesin antigen coupled with a carrier particle. The antigen and. . .

IT Eubacteria
 (***ghosts*** , as carrier particles; immunogenic multivalent adhesins prepn. and use as vaccines)

IT Affinity chromatography
 Bacterial infection
 Gram-negative ***bacteria***
 Linking agents
 Pharmaceutical carriers
 Vaccines
 (immunogenic multivalent adhesins prepn. and use as vaccines)

AN 2007:1171779 CAPLUS <<LOGINID::20091202>>
DN 147:467781
TI Her-2/neu multi-peptide cancer vaccine
IN Zielinski, Christoph; Schreiner, Otto; Pehamberger, Hubert; Breiteneder, Heimo; Wiedermann, Ursula
PA Bio Life Science Forschungs- und Entwicklungsges.m.b.H., Austria
SO Eur. Pat. Appl., 26pp.
CODEN: EPXXDW

DT Patent

LA English

FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|----------|---|------|----------|-----------------|----------|
| PI | EP 1844788 | A1 | 20071017 | EP 2006-7834 | 20060413 |
| | R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU | | | | |
| | AU 2007237491 | A1 | 20071025 | AU 2007-237491 | 20070411 |
| | CA 2649013 | A1 | 20071025 | CA 2007-2649013 | 20070411 |
| | WO 2007118660 | A2 | 20071025 | WO 2007-EP3226 | 20070411 |
| | WO 2007118660 | A3 | 20071213 | | |
| | W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, US, UZ, VC, VN, ZA, ZM, ZW | | | | |
| | RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA | | | | |
| | EP 2004218 | A2 | 20081224 | EP 2007-724167 | 20070411 |
| | R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR | | | | |
| | US 20090269364 | A1 | 20091029 | US 2009-296738 | 20090403 |
| PRAI | EP 2006-7834 | A | 20060413 | | |
| | WO 2007-EP3226 | W | 20070411 | | |
| AB | A multi-peptide multipeptope vaccine against cancers assocd. with HER-2/neu oncogene overexpression is disclosed. The vaccine comprises a specific combination of peptides presenting different amino acids sequences that are present in the extracellular domain of HER-2/neu protein. The inventors demonstrate that the above vaccine is effective in preventing neu-expressing tumors and that the effect could be increased by co-administration of interleukin-12. Also, the vaccine could be administered as a mucosal vaccine without losing its high immunogenicity, which would be an attractive vaccine for tumors located at mucosal surfaces. | | | | |
| RE.CNT 8 | THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT | | | | |

IT Drug delivery systems
(***bacterial*** ***ghosts*** , vaccine carriers; her-2/neu multi-peptide cancer vaccine)
IT Interleukin 2
Interleukin 4
neu (***receptor***)

RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (her-2/neu multi-peptide cancer vaccine)
 IT Lactic acid ***bacteria***
 (mucosal adjuvants; her-2/neu multi-peptide cancer vaccine)

L7 ANSWER 7 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 2007:502112 CAPLUS <>LOGINID::20091202>>
 DN 146:480526

TI Psudomonas quinolone signal and c-diGMP and conjugate as mucosal adjuvant
 for vaccine preparation against infection, autoimmune disease,
 inflammation, allergy, cancer and for fertility control
 IN Ebensen, Thomas; Morr, Michael; Guzman, Carlos A.
 PA GBF Gesellschaft fuer Biotechnologische Forschung mbH, Germany
 SO Eur. Pat. Appl., 43pp.
 CODEN: EPXXDW

DT Patent
 LA English
 FAN.CNT 1

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|------|----------|-----------------|----------|
| PI EP 1782826 | A1 | 20070509 | EP 2005-24266 | 20051108 |
| R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU | | | | |
| AU 2006312688 | A1 | 20070518 | AU 2006-312688 | 20061108 |
| AU 2006312692 | A1 | 20070518 | AU 2006-312692 | 20061108 |
| CA 2624903 | A1 | 20070518 | CA 2006-2624903 | 20061108 |
| CA 2624905 | A1 | 20070518 | CA 2006-2624905 | 20061108 |
| WO 2007054279 | A2 | 20070518 | WO 2006-EP10693 | 20061108 |
| WO 2007054279 | A3 | 20070830 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW | | | | |
| RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GO, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA | | | | |
| WO 2007054283 | A2 | 20070518 | WO 2006-EP10699 | 20061108 |
| WO 2007054283 | A3 | 20070809 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW | | | | |
| RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GO, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA | | | | |
| EP 1959989 | A2 | 20080827 | EP 2006-806710 | 20061108 |

R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR
EP 1959990 A2 20080827 EP 2006-828961 20061108
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR
US 20080286296 A1 20081120 US 2008-92747 20080506
IN 2008MN00958 A 20080718 IN 2008-MN958 20080512
US 20090169609 A1 20090702 US 2008-92518 20080825
PRAI EP 2005-24266 A 20051108
WO 2006-EP10693 W 20061108
WO 2006-EP10699 W 20061108
OS MARPAT 146:480526
AB The present invention relates to new adjuvants and the uses in pharmaceutical compns., such as in vaccines. In particular, the present invention provides new compds. useful as adjuvants and/or immunomodulators for prophylactic and/or therapeutic vaccination in the treatment of infectious diseases, inflammatory diseases, autoimmune diseases, tumors, and allergies as well as for the control of fertility in human or animal populations. The compds. are particularly useful not only as systemic agents, but preferably as mucosal adjuvants. In addn., the invention relates to its uses as active ingredients in pharmaceutical compns.
RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

IT Glycoproteins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(CD40-L (antigen CD40 ***ligand***) ; Psudomonas quinolone signal and c-diGMP and conjugate as mucosal adjuvant for vaccine prepn. against infection, autoimmune disease, inflammation, allergy, and cancer and for fertility control)

IT Eubacteria
(***ghost*** ***bacteria*** ; Psudomonas quinolone signal and c-diGMP and conjugate as mucosal adjuvant for vaccine prepn. against infection, autoimmune disease, inflammation, allergy, and cancer and for fertility control)

L7 ANSWER 8 OF 68 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
AN 2007370158 EMBASE <>LOGINID::20091202>>
TI Recent advances in delivery systems for anti-HIV1 therapy.
AU Lanao, Jose M. (correspondence); Briones, Elsa; Colino, Clara I.
CS Department of Pharmacy and Pharmaceutical Technology, Faculty of Pharmacy, University of Salamanca, Salamanca, Spain.
SO Journal of Drug Targeting, (Jan 2007) Vol. 15, No. 1, pp. 21-36.
Refs: 155
ISSN: 1061-186X; E-ISSN: 1029-2330 CODEN: JDTAEH
PUI 772692962
CY United Kingdom
DT Journal; Article
FS 026 Immunology, Serology and Transplantation
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
039 Pharmacy
004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
LA English
SL English

- ED Entered STN: 24 Aug 2007
Last Updated on STN: 24 Aug 2007
- AB In the last years, different non-biological and biological carrier systems have been developed for anti-HIV1 therapy. Liposomes are excellent potential anti-HIV1 carriers that have been tested with drugs, antisense oligonucleotides, ribozymes and therapeutic genes. Nanoparticles and low-density lipoproteins (LDLs) are cell-specific transporters of drugs against macrophage-specific infections such as HIV1. Through a process of protein transduction, cell-permeable peptides of natural origin or designed artificially allow the delivery of drugs and genetic material inside the cell. Erythrocyte ***ghosts*** and ***bacterial*** ***ghosts*** are a promising delivery system for therapeutic peptides and HIV vaccines. Of interest are the advances made in the field of HIV gene therapy by the use of autologous haematopoietic stem cells and viral vectors for HIV vaccines. Although important milestones have been reached in the development of carrier systems for the treatment of HIV, especially in the field of gene therapy, further clinical trials are required so that the efficiency and safety of these new systems can be guaranteed in HIV patients.
- AB . . . cell-permeable peptides of natural origin or designed artificially allow the delivery of drugs and genetic material inside the cell. Erythrocyte ***ghosts*** and ***bacterial*** ***ghosts*** are a promising delivery system for therapeutic peptides and HIV vaccines. Of interest are the advances made in the field. . .
- CT Medical Descriptors:
- Adenovirus
anemia: SI, side effect
antiviral activity
article
bacterial membrane
biodegradability
bone marrow toxicity: SI, side effect
CD4+ T lymphocyte
cellular immunity
drug accumulation
drug blood level
drug delivery system
drug half life
drug receptor binding
drug safety
encapsulation
erythrocyte ghost
human
Human immunodeficiency virus 1
*Human immunodeficiency virus 1 infection: DT, drug therapy
humoral immunity
leukopenia: SI, side effect
liposomal gene delivery system
nonhuman
nonviral gene delivery. . .
2 methylpiperazine: PD, pharmacology
aciclovir: PR, pharmaceutics
aciclovir: PD, pharmacology
adefovir: PD, pharmacology
antisense oligonucleotide: PR, pharmaceutics
antisense oligonucleotide: PD, pharmacology
aplavirok: DV, drug development

aplavirok: PD, pharmacology
chemokine receptor CCR5 antagonist: DV, drug development
chemokine receptor CCR5 antagonist: PD, pharmacology
didanosine: CB, drug combination
didanosine: PR, pharmaceutics
DNA vaccine: PR, pharmaceutics
flucytosine: PR, pharmaceutics
flucytosine: PD, pharmacology
ganciclovir: PR, pharmaceutics
glutathione: CB, . . .

L7 ANSWER 9 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2006:1124271 CAPLUS <>LOGINID::20091202>>

DN 145:434372

TI Nanosized biological container composed of virus capsid proteins, filled with fluorescent, magnetic, x-ray absorbent, nucleotide components and manufacture thereof

IN Chen, Liaohai; Bader, Samuel D.; Hoffmann, Axel F.; Kay, Brian K.; Makowski, Lee

PA The University of Chicago, USA

SO U.S. Pat. Appl. Publ., 27 pp.

CODEN: USXXCO

DT Patent

LA English

FAN.CNT 1

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|----------------------|------|----------|-----------------|----------|
| PI US 20060240456 | A1 | 20061026 | US 2006-384792 | 20060320 |
| PRAI US 2005-664235P | P | 20050322 | | |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Nanosized biol. containers that are " ***ghosts*** " of viruses for which capsids are independent of their endogenous viral nucleic acid cores, provide nano-particles of uniform size, and known nos. of sites for attachments of ligands. These containers can be filled with a fluorescent, magnetic, x-ray absorbent, nucleotide components or a radioactive particle and used as nanoscale markers.

AB Nanosized biol. containers that are " ***ghosts*** " of viruses for which capsids are independent of their endogenous viral nucleic acid cores, provide nano-particles of uniform size, and. . .

IT Fusion proteins (chimeric proteins)

RL: BSU (Biological study, unclassified); DEV (Device component use); BIOL (Biological study); USES (Uses)
(capsid protein comprising a ***ligand*** ; nanosized biol. container composed of virus capsid proteins, filled with fluorescent, magnetic, x-ray absorbent, nucleotide components and manuf. thereof)

IT Antibodies and Immunoglobulins

RL: BSU (Biological study, unclassified); DEV (Device component use); BIOL (Biological study); USES (Uses)
(***ligand*** ; nanosized biol. container composed of virus capsid proteins, filled with fluorescent, magnetic, x-ray absorbent, nucleotide components and manuf. thereof)

IT ***Bacteriophage***

Buffers

Fluorescent substances

Magnetic particles

Phage display library

Virus

(nanosized biol. container composed of virus capsid proteins, filled with fluorescent, magnetic, x-ray absorbent, nucleotide components and manuf. thereof)

L7 ANSWER 10 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN DUPLICATE 1
AN 2006:510281 BIOSIS <>LOGINID::20091202>>
DN PREV200600513565
TI A mechanism of cell death involving an adenylyl cyclase/PKA signaling pathway is induced by the Cry1Ab toxin of *Bacillus thuringiensis*.
AU Zhang, Xuebin; Candas, Mehmet; Griko, Natalya B.; Taussig, Ronald; Bulla, Lee A. Jr. [Reprint Author]
CS Univ Texas, Dept Mol and Cell Biol, Richardson, TX 75083 USA
lee.bulla@utdallas.edu
SO Proceedings of the National Academy of Sciences of the United States of America, (JUN 27 2006) Vol. 103, No. 26, pp. 9897-9902.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
LA English
ED Entered STN: 4 Oct 2006
Last Updated on STN: 4 Oct 2006
AB Many pathogenic organisms and their toxins target host cell receptors, the consequence of which is altered signaling events that lead to aberrant activity or cell death. A significant body of literature describes various molecular and cellular aspects of toxins associated with ***bacterial*** invasion, colonization, and host cell disruption. However, there is little information on the molecular and cellular mechanisms associated with the insecticidal action of *Bacillus thuringiensis* (Bt) Cry toxins. Recently, we reported that the Cry1Ab toxin produced by Bt kills insect cells by activating a Mg²⁺-dependent cytotoxic event upon binding of the toxin to its ***receptor*** BT-R-1. Here we show that binding of Cry toxin to BT-R1 provokes cell death by activating a previously undescribed signaling pathway involving stimulation of G protein (G(alpha s)) and adenylyl cyclase, increased cAMP levels, and activation of protein kinase A. Induction of the adenylyl cyclase/protein kinase A pathway is manifested by sequential cytological changes that include membrane blebbing, appearance of ***ghost*** nuclei, cell swelling, and lysis. The discovery of a toxin-induced cell death pathway specifically linked to BT-R-1 in insect cells should provide insights into how insects evolve resistance to Bt and into the development of new, safer insecticides.
AB. . . aberrant activity or cell death. A significant body of literature describes various molecular and cellular aspects of toxins associated with ***bacterial*** invasion, colonization, and host cell disruption. However, there is little information on the molecular and cellular mechanisms associated with the. . . toxin produced by Bt kills insect cells by activating a Mg²⁺-dependent cytotoxic event upon binding of the toxin to its ***receptor*** BT-R-1. Here we show that binding of Cry toxin to BT-R1 provokes cell death by activating a previously undescribed signaling. . . Induction of the adenylyl cyclase/protein kinase A pathway is manifested by sequential cytological changes that include membrane blebbing, appearance of ***ghost*** nuclei, cell swelling, and lysis. The discovery of a toxin-induced cell death pathway specifically linked to BT-R-1 in insect cells. . .
IT . . .
Biochemicals
magnesium ion; cyclic AMP; Cry1Ab toxin: toxin; PKA [protein kinase A]:

signaling; adenylyl cyclase [EC 4.6.1.1]: signaling; BT-R-1 [Bt
receptor]

ORGN Classifier
Endospore-forming Gram-Positives 07810
Super Taxa
Eubacteria; ***Bacteria*** ; Microorganisms
Organism Name
Bacillus thuringiensis (species)
Taxa Notes
Bacteria , Eubacteria, Microorganisms

ORGN Classifier
Insecta 75300
Super Taxa
Arthropoda; Invertebrata; Animalia
Organism Name
H5 cell line (cell_line): insect cells
Taxa. . .

L7 ANSWER 11 OF 68 CABAB COPYRIGHT 2009 CABI on STN
AN 2008:83068 CABAB <>LOGINID::20091202>>
DN 20063203692
TI Advances in vaccine development against enterohemorrhagic Escherichia coli
O157:H7
AU Liu YanQing; Mao XuHu; Zou QuanMing; Liu, Y. Q.; Mao, X. H.; Zou, Q. M.
CS Clinical Microbiology and Immunology, The Third Medical University of PLA,
Chongqing 400038, China. mxh95xy@mail.tmmu.com.cn
SO Chinese Journal of Zoonoses, (2006) Vol. 22, No. 10, pp. 998-1000. 23 ref.
Publisher: Editorial Committee of Chinese Journal of Zoonoses, Health and
Anti-epidemic Station of Fujian Province. Fuzhou
ISSN: 1002-2694
URL: <http://www.zgrsghbzz.periodicals.net.cn>

CY China
DT Journal
LA Chinese
ED Entered STN: 5 May 2008
Last Updated on STN: 5 May 2008
AB Vaccine related protective antigens of enterohemorrhagic Escherichia coli
O157:H7 include adhesion antigens (e.g. intimin, translocated intimin
receptor and type III secretion system related protein EspA) and
toxic antigens. Polysaccharide vaccine, subunit vaccine, transgenic plant
vaccine and ***bacterial*** ***ghost*** vaccine have been
developed. Some vaccines have already been put into clinical trials.
AB Vaccine related protective antigens of enterohemorrhagic Escherichia coli
O157:H7 include adhesion antigens (e.g. intimin, translocated intimin
receptor and type III secretion system related protein EspA) and
toxic antigens. Polysaccharide vaccine, subunit vaccine, transgenic plant
vaccine and ***bacterial*** ***ghost*** vaccine have been
developed. Some vaccines have already been put into clinical trials.
BT Escherichia; Enterobacteriaceae; Gracilicutes; ***bacteria*** ;
prokaryotes

L7 ANSWER 12 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2005:120755 CAPLUS <>LOGINID::20091202>>
DN 142:225686
TI Sealing of ***bacterial*** ***ghosts*** for drug delivery using
membrane vesicles and affinity ***ligand*** interactions
IN Lubitz, Werner

PA Austria
 SO PCT Int. Appl., 37 pp.
 CODEN: PIXXD2
 DT Patent
 LA German
 FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------|---|------|----------|------------------|----------|
| PI | WO 2005011713 | A1 | 20050210 | WO 2004-EP8790 | 20040805 |
| | W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| DE | 10335796 | A1 | 20050303 | DE 2003-10335796 | 20030805 |
| AU | 2004260620 | A1 | 20050210 | AU 2004-260620 | 20040805 |
| AU | 2004260620 | B2 | 20080124 | | |
| CA | 2534612 | A1 | 20050210 | CA 2004-2534612 | 20040805 |
| EP | 1656149 | A1 | 20060517 | EP 2004-763831 | 20040805 |
| | R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK | | | | |
| NZ | 545232 | A | 20081224 | NZ 2004-545232 | 20040805 |
| US | 20060286126 | A1 | 20061221 | US 2006-567426 | 20060516 |
| PRAI | DE 2003-10335796 | A | 20030805 | | |
| | WO 2004-EP8790 | W | 20040805 | | |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The invention relates to method for producing sealed ***bacterial***
 ghosts using the specific interaction between partners of a binding pair. The ***ghosts*** can be loaded with therapeutically useful substances and used as carriers. The inventive sealed ***ghosts*** can be used in medicine, agriculture, and biotechnol. ***Ghosts*** are formed by inducing expression of the E gene, which causes membrane lysis. The ***ghosts*** are then derivatized with a member of a binding pair, e.g. ***biotin***, or a streptavidin-binding peptide. Biotinylation may be via an enzymic biotinylation site incorporated into the E gene product. The derivatized ***ghosts*** are then mixed with lipid vesicles present the other member of the binding pair, e.g. streptavidin. The interaction results in the binding of the lipid vesicles to the ***ghosts***. Sealed ***ghosts*** can be captured using the ***ligand*** immobilized on a suitable carrier.

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions
 AB The invention relates to a method for producing sealed ***bacterial***
 ghosts using the specific interaction between partners of a binding pair. The ***ghosts*** can be loaded with therapeutically useful substances and used as carriers. The inventive sealed ***ghosts*** can be used in medicine, agriculture, and biotechnol. ***Ghosts*** are formed by inducing expression of the E gene, which causes membrane lysis. The ***ghosts*** are then derivatized with a

member of a binding pair, e.g. ***biotin***, or a streptavidin-binding peptide. Biotinylation may be via an enzymic biotinylation site incorporated into the E gene product. The derivatized ***ghosts*** are then mixed with lipid vesicles present the other member of the binding pair, e.g. streptavidin. The interaction results in the binding of the lipid vesicles to the ***ghosts***. Sealed ***ghosts*** can be captured using the ***ligand*** immobilized on a suitable carrier.

ST ***bacteria*** membrane ***ghost*** sealing lipid vesicle affinity interaction; membrane ***biotin*** vesicle streptavidin
 bacteria ***ghost*** sealing

IT Gene, microbial
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
 (E; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Drug delivery systems
 (***bacterial*** ***ghosts*** as; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Transformation, genetic
 (***bacterial*** ***ghosts*** for delivery of nucleic acids in; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Agrochemicals
Drugs
Dyes
Organelle
 (***bacterial*** ***ghosts*** for delivery of; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Nucleic acids
RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (***bacterial*** ***ghosts*** for delivery of; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Protein motifs
 (biotinylation, lysis proteins contg.; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Protoplast and Spheroplast
 (cell ***ghost*** ; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Virion structure
 (envelope, sealing of membrane ***ghosts*** with; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Antibodies and Immunoglobulins
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
 (fragments, in affinity binding of membrane vesicles to ***bacterial*** ***ghosts*** ; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Agglutinins and Lectins
Antibodies and Immunoglobulins
Avidins

Carbohydrates, biological studies
Haptens
Receptors
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(in affinity binding of membrane vesicles to ***bacterial***
ghosts ; sealing of ***bacterial*** ***ghosts*** for
drug delivery using membrane vesicles and affinity ***ligand***
interactions)

IT Eubacteria
(membrane ***ghosts*** ; sealing of ***bacterial***
ghosts for drug delivery using membrane vesicles and affinity
ligand interactions)

IT Proteins
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(membrane, incorporation into ***bacterial*** ***ghosts*** of;
sealing of ***bacterial*** ***ghosts*** for drug delivery using
membrane vesicles and affinity ***ligand*** interactions)

IT Immobilization, molecular or cellular
(of ***bacterial*** ***ghosts*** ; sealing of ***bacterial***
ghosts for drug delivery using membrane vesicles and affinity
ligand interactions)

IT Gram-negative ***bacteria***
(prep. of membrane ***ghosts*** from; sealing of ***bacterial***
ghosts for drug delivery using membrane vesicles and affinity
ligand interactions)

IT Agriculture and Agricultural chemistry
Biotechnology
Medicine
(sealing of ***bacterial*** ***ghosts*** for drug delivery
using membrane vesicles and affinity ***ligand*** interactions)

IT Liposomes
(sealing of membrane ***ghosts*** with; sealing of
bacterial ***ghosts*** for drug delivery using membrane
vesicles and affinity ***ligand*** interactions)

IT Lipids, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(vesicles, sealing of membrane ***ghosts*** with; sealing of
bacterial ***ghosts*** for drug delivery using membrane
vesicles and affinity ***ligand*** interactions)

IT 58-85-5D, ***Biotin*** , analogs, conjugates with proteins 9013-20-1,
Streptavidin
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(in affinity binding of membrane vesicles to ***bacterial***
ghosts ; sealing of ***bacterial*** ***ghosts*** for
drug delivery using membrane vesicles and affinity ***ligand***
interactions)

IT 842177-75-7 842177-76-8 842177-77-9 842177-78-0 842177-79-1
842177-80-4
RL: PRP (Properties)
(unclaimed nucleotide sequence; sealing of ***bacterial***
ghosts for drug delivery using membrane vesicles and affinity
ligand interactions)

IT 842138-49-2

RL: PRP (Properties)
(unclaimed sequence; sealing of ***bacterial*** ***ghosts***
for drug delivery using membrane vesicles and affinity ***ligand***
interactions)

L7 ANSWER 13 OF 68 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN DUPLICATE 2

AN 2005434767 EMBASE <>LOGINID::20091202>>

TI A novel fluorescent probe: Europium complex hybridized T7 phage.

AU Liu, Chin-Mei; Jin, Qiaoling; Sutton, April; Chen, Liaohai
(correspondence)

CS Biosciences Division, Argonne National Laboratory, 9700 South Cass Avenue, Argonne, IL 60439, United States. lhchen@anl.gov

SO Bioconjugate Chemistry, (Sep 2005) Vol. 16, No. 5, pp. 1054-1057.

Refs: 16

CY ISSN: 1043-1802 CODEN: BCCHE

DT United States

Journal; Article

FS 023 Nuclear Medicine
037 Drug Literature Index
039 Pharmacy
004 Microbiology: Bacteriology, Mycology, Parasitology and Virology

LA English

SL English

ED Entered STN: 27 Oct 2005
Last Updated on STN: 27 Oct 2005

AB We report on the creation of a novel fluorescent probe of europium-complex hybridized T7 phage. It was made by filling a ***ligand*** -displayed T7 ***ghost*** phage with a fluorescent europium complex particle. The structure of the hybridized phage, which contains a fluorescent inorganic core surrounded by a ***ligand*** -displayed capsid shell, was confirmed by electron microscope, energy-dispersive X-ray analysis (EDX), bioassays, and fluorescence spectrometer. More importantly, as a benefit of the phage display technology, the hybridized phage has the capability to integrate an affinity reagent against virtually any target molecules. The approach provides an original method to fluorescently "tag" a bioligand and/or to "biofunctionalize" a fluorophore particle. By using other types of materials such as radioactive or magnetic particles to fill the ***ghost*** phage, we envision that the hybridized phages represent a new class of fluorescent, magnetic, or radioprobes for imaging and bioassays and could be used both in vitro and in vivo. .COPYRGT. 2005 American Chemical Society.

AB . . . report on the creation of a novel fluorescent probe of europium-complex hybridized T7 phage. It was made by filling a ***ligand*** -displayed T7 ***ghost*** phage with a fluorescent europium complex particle. The structure of the hybridized phage, which contains a fluorescent inorganic core surrounded by a ***ligand*** -displayed capsid shell, was confirmed by electron microscope, energy-dispersive X-ray analysis (EDX), bioassays, and fluorescence spectrometer. More importantly, as a benefit. . . to "biofunctionalize" a fluorophore particle. By using other types of materials such as radioactive or magnetic particles to fill the ***ghost*** phage, we envision that the hybridized phages represent a new class of fluorescent, magnetic, or radioprobes for imaging and bioassays. . .

CT Medical Descriptors:
article

****bacteriophage T7****
bioassay
complex formation
electron microscopy
fluorescence
hybridization
imaging
spectrofluorometry
X ray analysis
*europium: DV, drug development
*europium: PR, pharmaceutics
*fluorescent dye: DV, drug development
*fluorescent dye: PR, pharmaceutics
lanthanide: DV, drug development
lanthanide: PR, pharmaceutics
 ligand: DV, drug development
 ligand: PR, pharmaceutics
radioactive material: DV, drug development
radioactive material: PR, pharmaceutics

L7 ANSWER 14 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN DUPLICATE 3
AN 2005:112300 BIOSIS <>LOGINID::20091202>>
DN PREV200500114316
TI Translocation of histone proteins across lipid bilayers and mycoplasma
membranes.
AU Rosenbluh, Joseph; Hariton-Gazal, Elana; Dagan, Arie; Rottem, Shlomo;
Graessmann, Adolf; Loyter, Abraham [Reprint Author]
CS Alexander Silberman Inst Life SciDept Biol Chem, Hebrew Univ Jerusalem,
IL-91904, Jerusalem, Israel
loyter@mail.ls.huji.ac.il
SO Journal of Molecular Biology, (January 14 2005) Vol. 345, No. 2, pp.
387-400. print.
ISSN: 0022-2836 (ISSN print).
DT Article
LA English
ED Entered STN: 23 Mar 2005
Last Updated on STN: 23 Mar 2005
AB We show that the three core histones H2A, H3 and H4 can transverse lipid
bilayers of large unilamellar vesicles (LUVs) and multilamellar vesicles
(MLVs). In contrast, the histone H2B, although able to bind to the
liposomes, fails to penetrate the unilamellar and the multilamellar
vesicles. Translocation across the lipid bilayer was determined using
biotin -labeled histones and an ELISA-based system. Following
incubation with the liposomes, external membrane-bound ***biotin***
molecules were neutralized by the addition of ***avidin*** .
Penetrating ***biotin*** -histone conjugates were exposed by Triton
treatment of the neutralized liposomes. The intraliposomal
biotin -histone conjugates, in contrast to those attached only to
the external surface, were attached to the detergent lysed lipid
molecules. Thus, biotinylated histone molecules that were exposed only
following detergent treatment of the liposomes were considered to be
located at the inner leaflet of the lipid bilayers. The penetrating
histone molecules failed to mediate translocation of BSA molecules
covalently attached to them. Translocation of the core histones,
including H2B, was also observed across mycoplasma cell membranes. The
extent of this translocation was inversely related to the degree of

membrane cholesterol. The addition of cholesterol also reduced the extent of histone penetration into the MLVs. Although able to bind biotinylated histones, human erythrocytes, erythrocyte ***ghosts*** and Escherichia coli cells were impermeable to them. Based on the present and previous data histones appear to be characterized by the same features that characterize cell penetrating peptides and proteins (CPPs). Copyright 2004 Elsevier Ltd. All rights reserved.

AB. . . to the liposomes, fails to penetrate the unilamellar and the multilamellar vesicles. Translocation across the lipid bilayer was determined using ***biotin*** -labeled histones and an ELISA-based system. Following incubation with the liposomes, external membrane-bound ***biotin*** molecules were neutralized by the addition of ***avidin***. Penetrating ***biotin*** -histone conjugates were exposed by Triton treatment of the neutralized liposomes. The intra-liposomal ***biotin*** -histone conjugates, in contrast to those attached only to the external surface, were attached to the detergent lysed lipid molecules. Thus, . . . cholesterol also reduced the extent of histone penetration into the MLVs. Although able to bind biotinylated histones, human erythrocytes, erythrocyte ***ghosts*** and Escherichia coli cells were impermeable to them. Based on the present and previous data histones appear to be characterized. . .

IT . . .

IT Parts, Structures, & Systems of Organisms
erythrocyte: blood and lymphatics

IT Chemicals & Biochemicals
H2A; H3 histone; H4 histone; ***avidin*** ; cell-penetrating peptides; histone protein; lipid bilayer; liposome; membrane-bound protein

ORGN Classifier

Enterobacteriaceae 06702

Super Taxa

Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria ; Microorganisms

Organism Name

Escherichia coli (species)

Taxa Notes

Bacteria , Eubacteria, Microorganisms

L7 ANSWER 15 OF 68 SCISEARCH COPYRIGHT (c) 2009 The Thomson Corporation on STN

AN 2005:77370 SCISEARCH <>LOGINID::20091202>>

GA The Genuine Article (R) Number: 883WX

TI Rational design of vaccination strategies to promote antigen entry into the MHC class I-restricted presentation pathway

AU Guzman C A (Reprint)

CS GBF German Res Ctr Biotechnol, Div Microbiol, Vaccine Res Grp, Mascheroder Weg 1, D-38124 Braunschweig, Germany (Reprint)

AU Becker P D

CS GBF German Res Ctr Biotechnol, Div Microbiol, Vaccine Res Grp, D-38124 Braunschweig, Germany
E-mail: cag@gbf.de

CY A Germany

SO TRANSFUSION MEDICINE AND HEMOTHERAPY, (2004) Vol. 31, No. 6, pp. 398-411.
ISSN: 1660-3796.

PB KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.

DT General Review; Journal

LA English

REC Reference Count: 180
ED Entered STN: 27 Jan 2005
Last Updated on STN: 27 Jan 2005
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Cytotoxic CD8+ T lymphocytes (CTLs) constitute one of the main effector mechanisms against tumors and viral infections. CTLs specifically recognize short peptides (8 - 10 residues long) displayed on the surface of 'target' cells, which result from the processing of foreign or abnormal proteins (e. g. virus and tumor proteins) and are bound to major histocompatibility complex (MHC) class I molecules. Virtually all nucleated cells display on their surface fragments of intracellularly produced polypeptides. When there are signs of invasion or transformation, CTLs take control of the situation by destroying these 'labeled' target cells. This is an extremely efficient mechanism. However, the efficient differentiation of naive CD8+ T cells into CTLs is a limiting prerequisite. To achieve this differentiation, dendritic cells (DCs) are critical since only these professional antigen-presenting cells (APCs) can provide not only the peptide presented onto the MHC class I molecules but also the costimulatory signals required for this activation. To this end, DCs take up antigens and degrade them into peptides which are loaded on MHC class I and presented onto the surface to prime specific T lymphocytes. In this review, we summarize the current knowledge on the mechanisms used by professional APCs in the processing and presentation of endogenous and exogenous antigens in the context of MHC class I molecules (i.e. priming and cross-priming). We will also discuss new vaccination strategies that take advantage of these physiological mechanisms in order to improve the elicitation of cytotoxic responses to eliminate intracellular pathogens and tumors.

STP KeyWords Plus (R): COMPLEX CLASS-I; RECOMBINANT LISTERIA-MONOCYTOGENES; CYTOTOXIC T-LYMPHOCYTES; DENDRITIC CELL MATURATION; ***RECEPTOR***-MEDIATED ENDOCYTOSIS; PROTEIN-CHAPERONED PEPTIDES; EPITOPE PRECURSOR PEPTIDES; EXOGENOUS SOLUBLE-ANTIGEN; ***BACTERIAL*** ***GHOST*** SYSTEM; TOLL-LIKE ***RECEPTOR*** -9

L7 ANSWER 16 OF 68 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
AN 2003192797 EMBASE <<LOGINID::20091202>>
TI ***Bacterial*** ***ghosts*** as carrier and targeting systems for mucosal antigen delivery.
AU Jalava, Katri (correspondence); Lubitz, Werner
CS BIRD-C GmbH and CoKEG, Schoenborngasse 12, A-1080 Wien, Austria.
jalava@bird-c.com
AU Eko, Francis O.
CS Department of Microbiology, Morehouse School of Medicine, Atlanta, GA, United States.
AU Riedmann, Eva; Lubitz, Werner
CS Inst. of Microbiology and Genetics, University of Vienna, Vienna, Austria.
SO Expert Review of Vaccines, (Feb 2003) Vol. 2, No. 1, pp. 45-51.
Refs: 42
ISSN: 1476-0584 CODEN: ERVXAX
CY United Kingdom
DT Journal; General Review; (Review)
FS 026 Immunology, Serology and Transplantation
037 Drug Literature Index
039 Pharmacy
004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
LA English

SL English
ED Entered STN: 29 May 2003
Last Updated on STN: 29 May 2003

AB The application of new strategies to develop effective vaccines is essential in modern medicine. The ***bacterial*** ***ghost*** system is a novel vaccine delivery system endowed with intrinsic adjuvant properties. ***Bacterial*** ***ghosts*** are nonliving Gram-negative ***bacterial*** cell envelopes devoid of cytoplasmic contents while maintaining their cellular morphology and native surface antigenic structures including bioadhesive properties. They are produced by PhIX174 protein E-mediated lysis of Gram-negative ***bacteria***. The intrinsic adjuvant properties of ***bacterial*** ***ghost*** preparations enhance immune responses against envelope-bound antigens, including T-cell activation and mucosal immunity. Since native and foreign antigens can be expressed in the envelope complex of ***ghosts*** before E- mediated lysis, multiple antigens of various origin can be presented to the immune system simultaneously. In addition, the extended ***bacterial*** ***ghost*** system represents a platform technology for specific targeting of DNA-encoded antigens to primary antigen-presenting cells. The potency, safety and relatively low production cost of ***bacterial*** ***ghosts*** offer a significant technical advantage, especially when used as combination vaccines.

TI ***Bacterial*** ***ghosts*** as carrier and targeting systems for mucosal antigen delivery.

AB The application of new strategies to develop effective vaccines is essential in modern medicine. The ***bacterial*** ***ghost*** system is a novel vaccine delivery system endowed with intrinsic adjuvant properties. ***Bacterial*** ***ghosts*** are nonliving Gram-negative ***bacterial*** cell envelopes devoid of cytoplasmic contents while maintaining their cellular morphology and native surface antigenic structures including bioadhesive properties. They are produced by PhIX174 protein E-mediated lysis of Gram-negative ***bacteria***. The intrinsic adjuvant properties of ***bacterial*** ***ghost*** preparations enhance immune responses against envelope-bound antigens, including T-cell activation and mucosal immunity. Since native and foreign antigens can be expressed in the envelope complex of ***ghosts*** before E- mediated lysis, multiple antigens of various origin can be presented to the immune system simultaneously. In addition, the extended ***bacterial*** ***ghost*** system represents a platform technology for specific targeting of DNA-encoded antigens to primary antigen-presenting cells. The potency, safety and relatively low production cost of ***bacterial*** ***ghosts*** offer a significant technical advantage, especially when used as combination vaccines.

CT Medical Descriptors:
aerosol
antigen expression
antigen presentation
antigen presenting cell
 ****bacterial infection: DT, drug therapy***
 bacterial infection: PC, prevention
 bacterial membrane
 bacterial strain
cell structure
chlamydiasis: DT, drug therapy
chlamydiasis: PC, prevention

cholera: DT, drug therapy
cholera: PC, prevention
cytoplasm
*drug delivery system
fertility
gene expression
 Gram negative bacterium
human
immune response
immune system
immunization
lysis
mucosal immunity
nonhuman
priority journal
review
T lymphocyte activation
aluminum potassium sulfate: PR, pharmaceutics
antigen: PR, pharmaceutics
 bacterial antigen: PR, pharmaceutics
 ****bacterial vaccine: DT, drug therapy***
 ****bacterial vaccine: PR, pharmaceutics***
chlamydia vaccine: DT, drug therapy
chlamydia vaccine: PR, pharmaceutics
cholera vaccine: DT, drug therapy
cholera vaccine: PO, oral drug administration
cholera vaccine: . . DT, drug therapy
contraceptive vaccine: NA, intranasal drug administration
contraceptive vaccine: PO, oral drug administration
contraceptive vaccine: PR, pharmaceutics
DNA: PR, pharmaceutics
Freund adjuvant: PR, pharmaceutics
 lectin: PR, pharmaceutics
lipid: PR, pharmaceutics
liposome: PR, pharmaceutics
muramyl dipeptide: PR, pharmaceutics
naked DNA: PR, pharmaceutics
polyethyleneimine: PR, pharmaceutics
polylysine: PR, pharmaceutics
polymer: PR, pharmaceutics
protein
protein e
toxin: PR, . . .

L7 ANSWER 17 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN
AN 2002:597026 BIOSIS <>LOGINID::20091202>>
DN PREV200200597026
TI Immunologic basis for the protective efficacy of Chlamydia vaccines.
AU Igietseme, J. U. [Reprint author]; Eko, F. O. [Reprint author]; Ananaba,
G. A.; Moore, T. [Reprint author]; McMillan, L. [Reprint author]; Ramey,
K. [Reprint author]; Jones, M. [Reprint author]; Zuzewicz, M. A.; He, Q.
[Reprint author]; Murdin, A.; Black, C.; Lyn, D. A. [Reprint author]
CS Morehouse School of Medicine, Atlanta, GA, USA
SO Abstracts of the General Meeting of the American Society for Microbiology,
(2002) Vol. 102, pp. 195. print.
Meeting Info.: 102nd General Meeting of the American Society for

- Microbiology. Salt Lake City, UT, USA. May 19-23, 2002. American Society for Microbiology.
ISSN: 1060-2011.
- DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
- LA English
- ED Entered STN: 20 Nov 2002
Last Updated on STN: 20 Nov 2002
- AB An efficacious vaccine that induces a sterilizing and long-term protective immunity is needed to control the ubiquitous oculogenital diseases caused by the obligate intracellular ***bacterium***, Chlamydia trachomatis. To define the cellular and molecular immunologic basis for the potency of a potentially efficacious vaccine against *C. trachomatis*, we analyzed a surrogate mouse model system of the genital infection. In this model system, an IL-10KO dendritic cell (DC)-based cellular vaccine confers a sterilizing, long-term protective anti-chlamydial genital immunity while two subunit vaccines (a chlamydial MOMP-ISCOMS preparation and *Vibrio cholerae* ***ghosts*** expressing chlamydial MOMP) induce a partial, short-term protection. The ability to confer protection correlates with the induction of genital mucosal Th1 response. Analysis of the kinetics of induction and maintenance of mucosal Th1 cells revealed that the DC-based regimen induced a greater (apprx5-fold) Th1 response than the MOMP-ISCOMS vaccine. Even at 200 days post immunization, the frequency of specific Th1 cells in the recipients of MOMP-ISCOMS were essentially reduced to the baseline naive mouse level; however, recipients of the DC-based cellular vaccine retained a relatively high Th1 response. The long-term protection from genital infection induced by the DC-based cellular vaccine was associated with the preservation of high frequency of Th1 cells, marked by the presence in the genital mucosa of mononuclear cells bearing the alpha1/beta2, alpha4/beta1, and alpha4/beta7 integrins, and specific antibodies, especially IgG2a. Finally, the dominant role of the Th1 cytokine, IFN-gamma, in protective anti-chlamydial immunity was revealed by the finding that the highly efficacious immune T cells from IL-10KO DC-based cellular vaccine immunized animals were ineffective in protecting IFN-gamma ***receptor*** knockout mice from the acute disease of genital chlamydial infection.
- AB. . . induces a sterilizing and long-term protective immunity is needed to control the ubiquitous oculogenital diseases caused by the obligate intracellular ***bacterium***, Chlamydia trachomatis. To define the cellular and molecular immunologic basis for the potency of a potentially efficacious vaccine against C. . . vaccine confers a sterilizing, long-term protective anti-chlamydial genital immunity while two subunit vaccines (a chlamydial MOMP-ISCOMS preparation and *Vibrio cholerae* ***ghosts*** expressing chlamydial MOMP) induce a partial, short-term protection. The ability to confer protection correlates with the induction of genital mucosal. . . finding that the highly efficacious immune T cells from IL-10KO DC-based cellular vaccine immunized animals were ineffective in protecting IFN-gamma ***receptor*** knockout mice from the acute disease of genital chlamydial infection.
- IT Major Concepts
 Immune System (Chemical Coordination and Homeostasis); Infection;
 Pharmacology
- IT Chemicals & Biochemicals
 bacterial vaccines: applications, development
- ORGN Classifier
 Chlamydiaceae 07121
 Super Taxa

Chlamydiales; Rickettsias and Chlamydias; Eubacteria; ***Bacteria***
 ; Microorganisms
 Organism Name
 Chlamydia spp.: pathogen
 Chlamydia trachomatis: pathogen
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms
 ORGN Classifier
 Muridae 86375
 Super Taxa
 Rodentia; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 mouse: animal model, host
 Taxa. . .

L7 ANSWER 18 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 2001:338673 CAPLUS <>LOGINID::20091202>>
 DN 134:350284
 TI Methods to screen microorganisms or gene libraries for products secreted
 from a cell
 IN Moeller, Soeren; Kongsbak, Lars; Kristensen, Hans-Henrik; Vind, Jesper;
 Pedersen, Henrik; Husum, Tommy Lykke
 PA Novozymes A/S, Den.
 SO PCT Int. Appl., 55 pp.
 CODEN: PIXXD2
 DT Patent
 LA English
 FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------|--|------|----------|-----------------|----------|
| PI | WO 2001032829 | A2 | 20010510 | WO 2000-DK566 | 20001010 |
| | WO 2001032829 | A3 | 20011213 | | |
| | W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW | | | | |
| | RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG | | | | |
| PRAI | AU 2000076460 | A | 20010514 | AU 2000-76460 | 20001010 |
| | DK 1999-1602 | A | 19991105 | | |
| | WO 2000-DK566 | W | 20001010 | | |
| AB | The invention describes methods for screening for products secreted from the cells, and provides methods to establish a correlation between the activity of the secreted product and the secreting cell. Accordingly in a first aspect the present invention relates to a method for screening a DNA library for DNA of interest comprising the steps of (a) creating host cells comprising the DNA library, (b) generating samples each comprising a host cell of step (a), (c) establishing a means for correlating of interest in a sample of the sample, (d) detg. which intensity interval of fluorescence indicates secretion in the sample when the correlating means of step (c) is used, (e) cultivating the samples under suitable conditions, and (f) selecting the samples exhibiting fluorescence within the intensity interval of step (d) using a fluorescence analyzer; wherein the host cell comprises DNA of interest. | | | | |

OSC.G 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

IT Erythrocyte
Macrophage
 (***ghost*** ; methods to screen microorganisms or gene libraries
 for products secreted from a cell)
IT Animal tissue culture
Antimicrobial agents
Aspergillus
Aspergillus nidulans
Aspergillus niger
Aspergillus oryzae
Bacillus (***bacterium*** genus)
Bacillus clausii
Bacillus licheniformis
Bacillus subtilis
 Bacteria (Eubacteria)
Carbon sources, microbial
Cell
Culture media
DNA sequences
Diffusion
Drugs
Encapsulation
Escherichia
Escherichia coli
Evolution
Films
Fluorescence
Fluorescent substances
Fluorometers
Fungi
Genomic library
Liposomes
Microorganism
Microspheres
Nucleic acid library
Samples
Secretion (process)
 (methods to screen microorganisms or gene libraries for products
 secreted from a cell)
IT 58-85-5, ***Biotin*** 81-88-9 2321-07-5, Fluorescein 9000-07-1,
Carrageenan 9000-69-5, Pectin 9000-92-4, Amylase 9003-05-8,
Polyacrylamide 9004-34-6, Cellulose, biological studies 9004-54-0,
Dextran, biological studies 9005-25-8, Starch, biological studies
9005-32-7, Alginic acid 9012-36-6, Agarose 9012-76-4, Chitosan
9013-20-1D, Streptavidin., Fluorescently labeled 51306-35-5
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES
(Uses)
 (methods to screen microorganisms or gene libraries for products
 secreted from a cell)

L7 ANSWER 19 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN DUPLICATE 4
AN 2001:301238 BIOSIS <>LOGINID::20091202>>
DN PREV200100301238

TI Glycophorin as a ***receptor*** for Escherichia coli alpha-hemolysin in erythrocytes.
AU Cortajarena, Aitziber L.; Goni, Felix M.; Ostolaza, Helena [Reprint author]
CS Unidad de Biofisica, Departamento de Bioquimica, Consejo Superior de Investigaciones Cientificas-UPV/EHU, Universidad del Pais Vasco/Euskal Herriko Unibertsitatea, Bilbao, 48080, Spain
gbzoseth@lg.ehu.es
SO Journal of Biological Chemistry, (April 20, 2001) Vol. 276, No. 16, pp. 12513-12519. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 27 Jun 2001
Last Updated on STN: 19 Feb 2002
AB Escherichia coli alpha-hemolysin (HlyA) can lyse both red blood cells (RBC) and liposomes. However, the cells are lysed at HlyA concentrations 1-2 orders of magnitude lower than liposomes (large unilamellar vesicles). Treatment of RBC with trypsin, but not with chymotrypsin, reduces the sensitivity of RBC toward HlyA to the level of the liposomes. Since glycophorin, one of the main proteins in the RBC surface, can be hydrolyzed by trypsin much more readily than by chymotrypsin, the possibility was tested of a specific binding of HlyA to glycophorin. With this purpose, a number of experiments were performed. (a) HlyA was preincubated with purified glycophorin, after which it was found to be inactive against both RBC and liposomes. (b) Treatment of RBC with an anti-glycophorin antibody protected the cells against HlyA lysis. (c) Immobilized HlyA was able to bind glycophorin present in a detergent lysate of RBC ***ghosts***. (d) Incorporation of glycophorin into pure phosphatidylcholine liposomes increased notoriously the sensitivity of the vesicles toward HlyA. (e) Treatment of the glycophorin-containing liposomes with trypsin reverted the vesicles to their original low sensitivity. The above results are interpreted in terms of glycophorin acting as a ***receptor*** for HlyA in RBC. The binding constant of HlyA for glycophorin was estimated, in RBC at sublytic HlyA concentrations, to be 1.5×10^{-9} M.
TI Glycophorin as a ***receptor*** for Escherichia coli alpha-hemolysin in erythrocytes.
AB . . . the cells against HlyA lysis. (c) Immobilized HlyA was able to bind glycophorin present in a detergent lysate of RBC ***ghosts***. (d) Incorporation of glycophorin into pure phosphatidylcholine liposomes increased notoriously the sensitivity of the vesicles toward HlyA. (e) Treatment of . . . reverted the vesicles to their original low sensitivity. The above results are interpreted in terms of glycophorin acting as a ***receptor*** for HlyA in RBC. The binding constant of HlyA for glycophorin was estimated, in RBC at sublytic HlyA concentrations, to. . .
ORGN Classifier
Enterobacteriaceae 06702
Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria ; Microorganisms
Organism Name
Escherichia coli
Taxa Notes
Bacteria , Eubacteria, Microorganisms

L7 ANSWER 20 OF 68 SCISEARCH COPYRIGHT (c) 2009 The Thomson Corporation on
STN

AN 2001:941959 SCISEARCH <>LOGINID::20091202>>

GA The Genuine Article (R) Number: 493YA

TI ***Bacterial*** ***ghosts*** as carrier and targeting systems

AU Lubitz W (Reprint)

CS Univ Vienna, Inst Microbiol & Genet, Dr Bohrgasse 9, A-1030 Vienna,
Austria (Reprint)
E-mail: werner.lubitz@univie.ac.at

CS Univ Vienna, Inst Microbiol & Genet, A-1030 Vienna, Austria

CYA Austria

SO EXPERT OPINION ON BIOLOGICAL THERAPY, (SEP 2001) Vol. 1, No. 5, pp.
765-771.

ISSN: 1471-2598.

PB INFORMA HEALTHCARE, TELEPHONE HOUSE, 69-77 PAUL STREET, LONDON EC2A 4LQ,
ENGLAND.

DT General Review; Journal

LA English

REC Reference Count: 48

ED Entered STN: 7 Dec 2001
Last Updated on STN: 24 Dec 2008

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB ***Bacterial*** ***ghosts*** are empty cell envelopes
originating from Gram-negative ***bacteria***. They have a natural
outer surface make-up which provides them with the original targeting
functions of the ***bacteria*** they are derived from and are thus
able to bind to and/or are taken up by specific cells or tissues of
animal, human or plant origin. The extended ***bacterial***
ghost system represents a platform technology for creating new
qualities in non-living carriers which can be used for the specific
targeting of drugs, DNA or other compounds to overcome toxic or
non-desired obstacles. Freeze dried ***bacterial*** ***ghosts***
are stable without the requirement of a cold chain and can be effectively
administered orally and aerogenically as drug carriers. The new system is
an alternative to liposomes and may have an advantage due to its higher
specificity for targeting specific tissues, its easy method of production
and its versatility in entrapping and packaging various compounds in
different compartments of the carriers.

TI ***Bacterial*** ***ghosts*** as carrier and targeting systems

AB ***Bacterial*** ***ghosts*** are empty cell envelopes
originating from Gram-negative ***bacteria***. They have a natural
outer surface make-up which provides them with the original targeting
functions of the ***bacteria*** they are derived from and are thus
able to bind to and/or are taken up by specific cells or tissues of
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ghost system represents a platform technology for creating new
qualities in non-living carriers which can be used for the specific
targeting of drugs, DNA or other compounds to overcome toxic or
non-desired obstacles. Freeze dried ***bacterial*** ***ghosts***
are stable without the requirement of a cold chain and can be effectively
administered orally and aerogenically as drug carriers.. . .

ST Author Keywords: ***bacterial*** ***ghost*** ; carrier and
targeting systems; drug delivery; synthetic gene delivery; therapy

STP KeyWords Plus (R): E-MEDIATED LYSIS; PHI-X174 GENE-E; ESCHERICHIA-COLI;
MANNOSE ***RECEPTOR*** ; PROTEIN-E; ENDOTHELIAL-CELLS; CANDIDATE
VACCINES; S-LAYERS; DENDRITIC CELLS; IN-SITU

L7 ANSWER 21 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN

AN 2002:223200 BIOSIS <>LOGINID::20091202>>

DN PREV200200223200

TI Functional P pilus-specific antibodies that block attachment of
bacteria to digalactosyl receptors.

AU Jian, L. [Reprint author]; Fusco, P. C. [Reprint author]

CS Baxter Healthcare Corporation, Columbia, MD, USA

SO Abstracts of the General Meeting of the American Society for Microbiology,
(2001) Vol. 101, pp. 340. print.
Meeting Info.: 101st General Meeting of the American Society for
Microbiology. Orlando, FL, USA. May 20-24, 2001. American Society of
Microbiology.
ISSN: 1060-2011.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 3 Apr 2002
Last Updated on STN: 3 Apr 2002

AB P pili from uropathogenic Escherichia coli have previously been used to
formulate vaccines to demonstrate protection against urinary tract
infections in mouse models. We have previously reported the role of P
pili in eliciting functionally active antibodies that block attachment of
purified pili to digalactosyl receptors on human erythrocyte
ghosts, independent of their tip adhesin, using an inhibition
ELISA-based method. In this study, an inhibition agglutination assay was
used to measure the inhibition of the pilated ***bacteria*** binding
to digalactosyl receptors on both human erythrocyte ***ghosts*** and
digalactosyl latex beads, using pilus-specific antisera, purified IgG, and
Fab fragments. Homologous and heterologous inhibition of
bacterial attachment was demonstrated with rabbit antisera
against F71, F72, F9, and F13 pili. Homologous endpoint inhibition titers for
digalactosyl latex beads were 12,800-25,600, which were 8-16 times higher
than with unrelated negative control sera. Heterologous inhibition titers
were 1-8 times higher than negative controls. ***Bacterial***
agglutination of human erythrocyte ***ghosts*** was inhibited by 50%
using homologous F71 antiserum diluted 1:38,400. In order to demonstrate
direct blocking of ***bacterial*** attachment independent of
bacterial agglutination, F71-specific Fab fragments were produced
and were shown to completely inhibit ***bacterial*** agglutination of
digalactosyl latex beads at 33 mug, with 70% inhibition occurring at 2.1
mug. In conclusion, evidence of adhesin-independent pilus-specific
blocking of attachment has been extended in vitro from purified pili to
piliated ***bacteria***.

TI Functional P pilus-specific antibodies that block attachment of
bacteria to digalactosyl receptors.

AB . . . of P pili in eliciting functionally active antibodies that block
attachment of purified pili to digalactosyl receptors on human erythrocyte
ghosts, independent of their tip adhesin, using an inhibition
ELISA-based method. In this study, an inhibition agglutination assay was
used to measure the inhibition of the pilated ***bacteria*** binding
to digalactosyl receptors on both human erythrocyte ***ghosts*** and
digalactosyl latex beads, using pilus-specific antisera, purified IgG, and
Fab fragments. Homologous and heterologous inhibition of
bacterial attachment was demonstrated with rabbit antisera
against

F71, F72, F9, and F13 pili. Homologous endpoint inhibition titers for digalactosyl latex. . . were 8-16 times higher than with unrelated negative control sera. Heterologous inhibition titers were 1-8 times higher than negative controls. ***Bacterial*** agglutination of human erythrocyte ***ghosts*** was inhibited by 50% using homologous F71 antiserum diluted 1:38,400. In order to demonstrate direct blocking of ***bacterial*** attachment independent of ***bacterial*** agglutination, F71-specific Fab fragments were produced and were shown to completely inhibit ***bacterial*** agglutination of digalactosyl latex beads at 33 mug, with 70% inhibition occurring at 2.1 mug. In conclusion, evidence of adhesin-independent pilus-specific blocking of attachment has been extended in vitro from purified pili to piliated ***bacteria***.

IT . . .

Infection

IT Parts, Structures, & Systems of Organisms
P pilus; erythrocyte: blood and lymphatics

IT Chemicals & Biochemicals
antibody; digalactosyl ***receptor*** ; immunoglobulin G

IT Miscellaneous Descriptors
bacterial agglutination; Meeting Abstract

ORGN Classifier
Enterobacteriaceae 06702

Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria ; Microorganisms

Organism Name

Escherichia coli: pathogen

Taxa Notes

Bacteria , Eubacteria, Microorganisms

ORGN Classifier
Hominidae 86215

Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

human

Taxa Notes

Animals, Chordates, . . .

L7 ANSWER 22 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2000:623585 CAPLUS <>LOGINID::20091202>>

DN 133:227782

TI ***Bacterial*** ***ghosts*** as carrier and targeting vehicles

IN Huter, Veronika; Lubitz, Werner

PA Austria

SO Ger. Offen., 10 pp.

CODEN: GWXXBX

DT Patent

LA German

FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|----|--|------|----------|------------------|----------|
| PI | DE 19909770 | A1 | 20000907 | DE 1999-19909770 | 19990305 |
| | CA 2370714 | A1 | 20000914 | CA 2000-2370714 | 20000303 |
| | WO 2000053163 | A1 | 20000914 | WO 2000-EP1906 | 20000303 |
| | W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, | | | | |

MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI,
 SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW
 RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
 DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
 CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
 EP 1158966 A1 20011205 EP 2000-912549 20000303
 EP 1158966 B1 20030611
 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
 IE, SI, LT, LV, FI, RO
 JP 2002538198 T 20021112 JP 2000-603652 20000303
 AT 242630 T 20030615 AT 2000-912549 20000303
 NZ 514408 A 20040130 NZ 2000-514408 20000303
 AU 778166 B2 20041118 AU 2000-34272 20000303
 PRAI DE 1999-19909770 A 19990305
 WO 2000-EP1906 W 20000303
AB Empty ***bacterial*** envelopes (***ghosts***), produced by controlled heterologous expression of a gene which effects a partial lysis of the cell membrane, are useful as carriers and targeting vehicles for active substances and markers. They may be administered via the natural infection pathways for pathogenic ***bacteria*** and are delivered specifically to the target tissues of the ***bacteria*** with high efficiency. Being empty, they can be loaded with active substances to a high degree. Agents which can be packaged in the ***ghosts*** include drugs, polypeptides, nucleic acids, agrochems., dyes, inks, and cosmetics; these may be immobilized by binding to specific receptors or binding sites incorporated into or anchored to the ***ghosts***. Thus, Escherichia coli NM522 cells were transformed simultaneously with plasmid pMLL (contg. phage .phi.X174 gene E encoding a transmembrane protein which induces leakage of the cell contents) and plasmid pAV1 (contg. the 54 5'-terminal codons of gene E fused in-frame to a coding sequence for the protease factor Xa recognition sequence and to 160 codons of the streptavidin gene). Expression of the streptavidin gene was induced with 3 mM IPTG, and expression of lysis protein E was subsequently induced by raising the temp. from 28.degree. to 42.degree.. Centrifugation of the cells and resuspension in distd. water resulted in immediate lysis, producing ***ghosts*** to which streptavidin was anchored. These ***ghosts*** strongly bound biotinylated alk. phosphatase, FITC- ***biotin*** , and other biotinylated agents.
 OSC.G 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)
 TI ***Bacterial*** ***ghosts*** as carrier and targeting vehicles
AB Empty ***bacterial*** envelopes (***ghosts***), produced by controlled heterologous expression of a gene which effects a partial lysis of the cell membrane, are useful as. . . carriers and targeting vehicles for active substances and markers. They may be administered via the natural infection pathways for pathogenic ***bacteria*** and are delivered specifically to the target tissues of the ***bacteria*** with high efficiency. Being empty, they can be loaded with active substances to a high degree. Agents which can be packaged in the ***ghosts*** include drugs, polypeptides, nucleic acids, agrochems., dyes, inks, and cosmetics; these may be immobilized by binding to specific receptors or binding sites incorporated into or anchored to the ***ghosts***. Thus, Escherichia coli NM522 cells were transformed simultaneously with plasmid pMLL (contg. phage .phi.X174 gene E encoding a transmembrane protein. . . the temp. from 28.degree. to 42.degree.. Centrifugation of the cells and resuspension in distd. water resulted in immediate lysis, producing ***ghosts*** to which streptavidin was anchored. These ***ghosts*** strongly bound biotinylated alk.

ST phosphatase, FITC- ***biotin*** , and other biotinylated agents.
bacteria ***ghost*** drug carrier targeting; streptavidin
bacteria ***ghost*** drug carrier

IT Gene, microbial
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(E, of phage .phi.X174, plasmid contg.; ***bacterial***
ghosts as carrier and targeting vehicles)

IT Polymers, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(active agent immobilization in matrix of; ***bacterial***
ghosts as carrier and targeting vehicles)

IT Diagnosis
(agents; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Agrochemicals
Anti-infective agents
Antitumor agents
Autoimmune disease
Bacteria (Eubacteria)

Cell membrane
Cytolysis
Drug targeting
Dyes
Gene therapy
Genetic markers
Gram-negative ***bacteria***
Gram-positive ***bacteria*** (Firmicutes)
Immobilization, biochemical
Vaccines
(***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Nucleic acids
Reporter gene
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Avidins
Polysaccharides, biological studies
Receptors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Drug delivery systems
(carriers; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT DNA
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(fluorescent-labeled; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Coliphage .phi.X174
(gene E protein of, lysis by; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Fatty acids, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(hydroxy, polymers; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Proteins, specific or class
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(***ligand*** -binding; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Aggregation
(matrix formation by; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Enzymes, uses
RL: CAT (Catalyst use); USES (Uses)
(matrix polmn. catalyzed by; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Encapsulation
(microencapsulation; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Plasmids
(streptavidin gene-contg.; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Fusion proteins (chimeric proteins)
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(streptavidin-contg.; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Protamines
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(sulfates; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT 146397-20-8
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(DNA labeled with; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT 25988-63-0, Poly-L-lysine hydrobromide 35013-72-0, ***Biotin***
N-hydroxysuccinimide ester
RL: RCT (Reactant); RACT (Reactant or reagent)
(***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT 9004-54-0, Dextran, biological studies 9013-20-1, Streptavidin
25104-18-1, Poly-L-lysine 38000-06-5, Poly-L-lysine
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT 9001-78-9D, biotinylated 25104-18-1D, Poly-L-lysine, biotinylated
38000-06-5D, Poly-L-lysine, biotinylated 134759-22-1
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(binding of, to streptavidin-contg. ***bacterial*** ***ghosts***
; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

L7 ANSWER 23 OF 68 SCISEARCH COPYRIGHT (c) 2009 The Thomson Corporation on
STN

AN 2000:710892 SCISEARCH <>LOGINID:20091202>>

GA The Genuine Article (R) Number: 354GJ

TI Interaction of Bartonella bacilliformis with human erythrocyte membrane

proteins
AU Hill E M (Reprint)
CS Meharry Med Coll, Sch Grad Studies, Dept Microbiol, Nashville, TN 37208 USA (Reprint)
AU Buckles E L
CS Meharry Med Coll, Sch Med, Nashville, TN 37208 USA
CYA USA
SO MICROBIAL PATHOGENESIS, (SEP 2000) Vol. 29, No. 3, pp. 165-174.
ISSN: 0882-4010.
PB ACADEMIC PRESS LTD, 24-28 OVAL RD, LONDON NW1 7DX, ENGLAND.
DT Article; Journal
LA English
REC Reference Count: 25
ED Entered STN: 2000
Last Updated on STN: 2000
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Intracellular invasion is an important aspect of Carrion's disease caused by *Bartonella bacilliformis*. Both the hematic and tissue phases of the disease involve the initial attachment of the organism to erythrocytes and endothelial cells, respectively. Using two different approaches, preliminary evidence is provided that *B. bacilliformis* interacts with multiple surface-exposed proteins on human erythrocytes. Utilizing Western blot analysis, it was demonstrated that the organism binds several biotinylated erythrocyte proteins with approximate molecular masses of 230, 210, 100, 83 and 44 kDa. There was enhanced *Bartonella* binding to the 44 kDa protein and binding to a 25 kDa protein following exposure of intact red cells to trypsin. Moreover, there was a complete abrogation of binding to these proteins following exposure of erythrocytes to sodium metaperiodate oxidation, indicating the significance of carbohydrate moieties in the interactions of *Bartonella* with the erythrocyte. In a second approach, similar binding proteins or putative receptors were identified when *Bartonella* was co-incubated with isolated membrane proteins from red cell ***ghosts***. A comparison of the molecular weights of these putative receptors with known erythrocyte proteins and their immunoreactivity to specific antisera suggested that the 230 and 210 kDa proteins are the alpha and beta subunits of spectrin; the 100 and 83 kDa proteins are band 3 protein and glycophorin A, respectively; and the 44 and 25 kDa proteins are the respective dimeric and monomeric forms of glycophorin B. Consistent with this notion was the binding of *Bartonella* to purified preparations of alpha and beta spectrin and glycophorin A/B. (C) 2000 Academic Press.
AB . . . approach, similar binding proteins or putative receptors were identified when *Bartonella* was co-incubated with isolated membrane proteins from red cell ***ghosts***. A comparison of the molecular weights of these putative receptors with known erythrocyte proteins and their immunoreactivity to specific antisera. . .
ST Author Keywords: *Bartonella bacilliformis*; ***bacterial*** adherence; human erythrocytes; glycophorin; band 3 protein; spectrin
STP KeyWords Plus (R): PLASMODIUM-FALCIPARUM; ENDOTHELIAL-CELLS; INVASION; HENSELAE; ***RECEPTOR*** ; ENTRY
L7 ANSWER 24 OF 68 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
AN 2000307384 EMBASE <>LOGINID::20091202>>
TI Characterization of p40/GPR69A as a peripheral membrane protein related to the lantibiotic synthetase component C.
AU Bauer, Hemma; Mayer, Herbert; Salzer, Ulrich; Prohaska, Rainer

(correspondence)

CS Institute of Medical Biochemistry, Department of Biochemistry, University of Vienna, Dr. Bohr-Gasse 9/3, A-1030 Vienna, Austria. prohaska@bch.univie.ac.at

AU Marchler-Bauer, Aron

CS National Center for Biotechnology Information, National Institutes of Health, Bethesda, MD 20894, United States.

SO Biochemical and Biophysical Research Communications, (18 Aug 2000) Vol. 275, No. 1, pp. 69-74.

Refs: 19

ISSN: 0006-291X CODEN: BBRCA9

CY United States

DT Journal; Article

FS 029 Clinical and Experimental Biochemistry

LA English

SL English

ED Entered STN: 21 Sep 2000

Last Updated on STN: 21 Sep 2000

AB The 40 kDa erythrocyte membrane protein p40/GPR69A, previously assigned to the G-protein-coupled ***receptor*** superfamily, was now identified by peptideantibodies and characterized as a loosely associated peripheral membrane protein. This result is in striking contrast to the proposed seven-transmembrane protein structure and function and therefore we wish to correct our previous proposal. p40 is located at the cytoplasmic side of the membrane and is neither associated with the cytoskeleton nor lipid rafts. Refined sequence analysis revealed that p40 is related to the LanC family of ***bacterial*** membrane-associated proteins which are involved in the biosynthesis of antimicrobial peptides. Therefore, we rename p40 to LanC-like protein 1 (LANCL1) and suggest that it may play a similar role as a peptide-modifying enzyme component in eukaryotic cells. (C) 2000 Academic Press.

AB The 40 kDa erythrocyte membrane protein p40/GPR69A, previously assigned to the G-protein-coupled ***receptor*** superfamily, was now identified by peptideantibodies and characterized as a loosely associated peripheral membrane protein. This result is in striking. . . associated with the cytoskeleton nor lipid rafts. Refined sequence analysis revealed that p40 is related to the LanC family of ***bacterial*** membrane-associated proteins which are involved in the biosynthesis of antimicrobial peptides. Therefore, we rename p40 to LanC-like protein 1 (LANCL1). . .

CT Medical Descriptors:

amino acid sequence

article

 erythrocyte ghost

*erythrocyte membrane

human

human cell

immunochemistry

priority journal

*protein analysis

protein degradation

protein structure

 ****G protein coupled receptor***

*lanthionine

*lantibiotic

*membrane protein

*peptide antibody

*synthetase

L7 ANSWER 25 OF 68 MEDLINE on STN
AN 1999456571 MEDLINE <>LOGINID::20091202>>
DN PubMed ID: 10525277
TI Maitotoxin-induced calcium influx in erythrocyte ***ghosts*** and rat glioma C6 cells, and blockade by gangliosides and other membrane lipids.
AU Konoki K; Hashimoto M; Murata M; Tachibana K
CS Department of Chemistry, School of Science, The University of Tokyo, Hongo, Bunkyo-ku, Tokyo 113-0033, Japan.
SO Chemical research in toxicology, (1999 Oct) Vol. 12, No. 10, pp. 993-1001. Journal code: 8807448. ISSN: 0893-228X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200001
ED Entered STN: 14 Jan 2000
Last Updated on STN: 14 Jan 2000
Entered Medline: 4 Jan 2000
AB Maitotoxin (MTX) at 0.3 nM elicited a 10-20-fold increase in the level of Ca(2+) influx in rat glioma C6 cells. At higher doses (3-30 nM), MTX induced marked Ca(2+) influx in human erythrocyte ***ghosts*** when monitored with the fluorescent dye Fura-2. Although the ***ghosts*** were not as susceptible to MTX as intact erythrocytes or other cell lines, Fura-2 experiments under various conditions suggested that the MTX-induced entry of ions into the ***ghosts*** was mediated by a mechanism similar to that reported for cells or tissues. These ***ghosts*** are the simplest system known to be sensitive to MTX and thus may be suitable for research on the direct action of MTX. Gangliosides GM1 and GM3, glycosphingolipids which have a sialic acid residue, strongly inhibited MTX-induced Ca(2+) influx in C6 cells, while the inhibitory action by asialo-GM1, which lacks a sialic acid residue, was somewhat weaker. Their inhibitory potencies were in the following order: GM1 (IC(50) approximately 2 microM) > GM3 (IC(50) approximately 5 microM) > asialo-GM1 (IC(50) approximately 20 microM). GM1 (3 microM) completely blocked MTX (30 nM)-induced Ca(2+) influx in human erythrocyte ***ghosts***. When C6 cells were pretreated with tunicamycin, an antibiotic which inhibits N-linked glycosylation, or concanavalin A, a ***lectin*** which exhibits a high affinity for cell-surface oligosaccharides, MTX-induced Ca(2+) influx was significantly potentiated. This suggests that removal of oligosaccharides from the cell surface by tunicamycin or capping of sugar chains on plasma membranes by concanavalin A can potentiate the action of MTX.
TI Maitotoxin-induced calcium influx in erythrocyte ***ghosts*** and rat glioma C6 cells, and blockade by gangliosides and other membrane lipids.
AB . . . Ca(2+) influx in rat glioma C6 cells. At higher doses (3-30 nM), MTX induced marked Ca(2+) influx in human erythrocyte ***ghosts*** when monitored with the fluorescent dye Fura-2. Although the ***ghosts*** were not as susceptible to MTX as intact erythrocytes or other cell lines, Fura-2 experiments under various conditions suggested that the MTX-induced entry of ions into the ***ghosts*** was mediated by a mechanism similar to that reported for cells or tissues. These ***ghosts*** are the simplest system known to be sensitive to MTX and thus may be suitable for research on the direct. . . microM) > asialo-GM1 (IC(50) approximately 20 microM). GM1 (3 microM) completely blocked MTX (30 nM)-induced Ca(2+) influx in human erythrocyte

ghosts . When C6 cells were pretreated with tunicamycin, an antibiotic which inhibits N-linked glycosylation, or concanavalin A, a ***lectin*** which exhibits a high affinity for cell-surface oligosaccharides, MTX-induced Ca(2+) influx was significantly potentiated. This suggests that removal of oligosaccharides. . .

CT Animals
 *** Anti-Bacterial Agents: PD, pharmacology***
*Brain Neoplasms: ME, metabolism
*Calcium: BL, blood
Calcium Radioisotopes: DU, diagnostic use
Concanavalin A: PD, pharmacology

CN 0 (Anti- ***Bacterial*** Agents); 0 (Calcium Radioisotopes); 0 (Fluorescent Dyes); 0 (Gangliosides); 0 (Marine Toxins); 0 (Membrane Lipids); 0 (Oxocins)

L7 ANSWER 26 OF 68 SCISEARCH COPYRIGHT (c) 2009 The Thomson Corporation on STN

AN 1999:828113 SCISEARCH <>LOGINID::20091202>>

GA The Genuine Article (R) Number: 250PR

TI ***Bacterial*** cell envelopes (***ghosts***) but not S-layers activate human endothelial cells (HUVECs) through sCD14 and LBP mechanism

AU Furst-Ladani S (Reprint)

CS Ludwig Boltzmann Inst Expt & Clin Traumatol, Donaueschingenstr 13, A-1200 Vienna, Austria (Reprint)

AU Redl H; Haslberger A; Lubitz W; Messner P; Sleytr U B; Schlag G

CS Ludwig Boltzmann Inst Expt & Clin Traumatol, A-1200 Vienna, Austria; Univ Vienna, Inst Microbiol & Genet, A-1090 Vienna, Austria; Univ Agr Sci, Ctr Ultrastruct Res, Vienna, Austria; Univ Agr Sci, Ludwig Boltzmann Inst Mol Nanotechnol, Vienna, Austria

CY Austria

SO VACCINE, (14 OCT 1999) Vol. 18, No. 5-6, pp. 440-448.

ISSN: 0264-410X.

PB ELSEVIER SCI LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, OXON, ENGLAND.

DT Article; Journal

LA English

REC Reference Count: 26

ED Entered STN: 1999

Last Updated on STN: 1999

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB ***Bacterial*** cell-envelopes (called ***ghosts***) and surface layers (S-layers) are discussed to be used as vaccines and/or adjuvants, consequently it is necessary to find out which immunomodulatory mediators are induced in human cells. The present work focuses on the effects of ***ghosts*** (*Escherichia coli* O26:B6), S-layers (*Bacillus stearothermophilus*) in comparison with LPS and antibiotic-inactivated whole ***bacteria*** (*E. coli* O26:B6) on human umbilical vein endothelial cells (HUVEC) with regard to the release of interleukin 6 (IL-6) and the expression of surface E-selectin and the role of lipopolysaccharide binding protein (LBP), soluble CD14 (sCD14) and serum for this activation.

Endothelial cells responded to ***ghosts*** , whole ***bacteria*** and LPS with IL-6 release up to 15000 pg/ml and surface E-selectin expression, while in contrast the response to S-layers with IL-6 release up to 500 pg/ml was very weak. Compared to LPS, 10-100-fold higher concentrations of ***bacterial*** ***ghosts*** and whole

bacteria were required to induce the cytokine synthesis and E-selectin expression. IL-6 release and E-selectin expression of HUVECs were reduced in the absence of serum and equivalent to unstimulated samples. We have also studied the role of CD14 and LBP for the activation of endothelial cells using antiCD14 and antilBP antibodies (Ab). AntiCD14 and antilBP Ab both inhibited IL-6 release and E-selectin expression in a dose dependent manner after stimulation with ***ghosts***, whole ***bacteria*** and LPS but had no effect on S-layers stimulated cells. AntiCD14 Ab inhibited more effectively than antilBP Ab. These findings suggest that ***bacterial*** ***ghosts*** but not: S-layers activate HUVECs through sCD14 and LBP dependent mechanisms. (C) 1999 Elsevier Science Ltd. All rights reserved.

TI ***Bacterial*** cell envelopes (***ghosts***) but not S-layers activate human endothelial cells (HUVECs) through sCD14 and LBP mechanism
AB ***Bacterial*** cell-envelopes (called ***ghosts***) and surface layers (S-layers) are discussed to be used as vaccines and/or adjuvants, consequently it is necessary to find out which immunomodulatory mediators are induced in human cells. The present work focuses on the effects of ***ghosts*** (*Escherichia coli* O26:B6), S-layers (*Bacillus stearothermophilus*) in comparison with LPS and antibiotic-inactivated whole ***bacteria*** (*E. coli* O26:B6) on human umbilical vein endothelial cells (HUVEC) with regard to the release of interleukin 6 (IL-6) and . . . and the role of lipopolysaccharide binding protein (LBP), soluble CD14 (sCD14) and serum for this activation.

Endothelial cells responded to ***ghosts***, whole ***bacteria*** and LPS with IL-6 release up to 15000 pg/ml and surface E-selectin expression, while in contrast the response to S-layers with IL-6 release up to 500 pg/ml was very weak. Compared to LPS, 10-100-fold higher concentrations of ***bacterial*** ***ghosts*** and whole ***bacteria*** were required to induce the cytokine synthesis and E-selectin expression. IL-6 release and E-selectin expression of HUVECs were reduced in. . . (Ab). AntiCD14 and antilBP Ab both inhibited IL-6 release and E-selectin expression in a dose dependent manner after stimulation with ***ghosts***, whole ***bacteria*** and LPS but had no effect on S-layers stimulated cells. AntiCD14 Ab inhibited more effectively than antilBP Ab. These findings suggest that ***bacterial*** ***ghosts*** but not: S-layers activate HUVECs through sCD14 and LBP dependent mechanisms. (C) 1999 Elsevier Science Ltd. All rights reserved.

ST Author Keywords: endotoxicity; ***bacterial*** ***ghosts*** ;
 S-layers; cytokines; HUVEC

STP KeyWords Plus (R): LIPOPOLYSACCHARIDE-BINDING-PROTEIN; SOLUBLE CD14;
 IN-VITRO; LPS; MACROPHAGES; INDUCTION; RESPONSES; ENDOTOXIN; ALLERGEN;
 RECEPTOR

L7 ANSWER 27 OF 68 MEDLINE on STN

AN 1999411196 MEDLINE <>LOGINID::20091202>>

DN PubMed ID: 10481582

TI [Binding of the antileukemia drug *Escherichia coli* L-asparaginase to the plasma membrane of normal human mononuclear cells]. Asociación de la droga antileucémica L-asparaginasa de *Escherichia coli* a la membrana plasmática de células mononucleares humanas normales.

AU Mercado-Vianco L; Arenas-Díaz G

CS Laboratorio de Fisiología Celular, Instituto de Biología, Universidad Católica de Valparaíso, Chile.

SO Sangre, (1999 Jun) Vol. 44, No. 3, pp. 204-9.

Journal code: 0404373. ISSN: 0036-4355.

CY Spain
DT (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA Spanish
FS Priority Journals
EM 199910
ED Entered STN: 14 Oct 1999
Last Updated on STN: 14 Oct 1999
Entered Medline: 7 Oct 1999

AB OBJECTIVE: To demonstrate that the enzyme L-asparaginase from Escherichia coli (EcA) binds to the plasma membranes of normal human lymphocytes and monocytes. MATERIAL AND METHODS: Lymphocytes and monocytes were isolated from heparinized blood samples which came from healthy volunteer donors. The cells were incubated with EcA to detect a possible binding of the enzyme to the mononuclear cells by indirect immunofluorescence using confocal microscopy. Meanwhile, ultracentrifugation was used to obtain the erythrocyte ***ghost*** microsomal fraction (P100) which was then analyzed by Western blotting to determine if EcA binds the lipid bilayer unspecifically. For the immunoassays, monospecific polyclonal antibodies were obtained from ascitic tumors developed in mice immunized with commercial L-asparaginase. RESULTS: EcA binds the lymphocyte and monocyte plasma membranes. In monocytes, there occurs a capping phenomenon, that is, the accumulation of fluorescent marker in one region. The image analyzer highlights it clearly at a depth of 3.8 microns. This binding would be unspecific, that is, there is no mediation of a specific ***receptor*** that binds EcA. This arises from the ability of the enzyme to bind to the membranes of erythrocyte ***ghost***, as evidenced by the ability of the molecule to associate with a hydrophobic medium. The antibodies against EcA obtained from ascitic tumors developed in mice do not show cross reactivity with Na+/K+ ATPase, aspartate aminotransferase, nor with extracts of blood cells, which would make it a specific tool for the detection of EcA in whole cells and in homogenates electrotransferred to nitrocellulose membranes. CONCLUSION: L-asparaginase from E. coli behaves as a lipoprotein due to its ability to insert itself into hydrophobic environments, in which it resembles an isozyme present in T. pyriformis. The binding of this enzyme to lymphocytes and monocytes, demonstrated in this work, would permit the modification of the antileukemic treatment injecting doses of EcA bound to patient's own isolated immune cells.

AB . . . the enzyme to the mononuclear cells by indirect immunofluorescence using confocal microscopy. Meanwhile, ultracentrifugation was used to obtain the erythrocyte ***ghost*** microsomal fraction (P100) which was then analyzed by Western blotting to determine if EcA binds the lipid bilayer unspecifically. For. . . at a depth of 3.8 microns. This binding would be unspecific, that is, there is no mediation of a specific ***receptor*** that binds EcA. This arises from the ability of the enzyme to bind to the membranes of erythrocyte ***ghost***, as evidenced by the ability of the molecule to associate with a hydrophobic medium. The antibodies against EcA obtained from. . .

CT . . . Agents: AD, administration & dosage
*Antineoplastic Agents: ME, metabolism
Ascites: IM, immunology
Asparaginase: AD, administration & dosage
*Asparaginase: ME, metabolism
*** Bacterial Proteins: AD, administration & dosage***

****Bacterial Proteins: ME, metabolism***
*Cell Membrane: ME, metabolism
Cross Reactions
Drug Carriers
Erythrocyte Membrane: ME, metabolism
Escherichia coli: EN, enzymology
Fluorescent. . . DE, drug effects
Lymphocytes: ME, metabolism
Membrane Lipids: ME, metabolism
Mice
Microscopy, Confocal
Monocytes: DE, drug effects
Monocytes: ME, metabolism
*** Receptor Aggregation***
Thromboembolism: CI, chemically induced
Thromboembolism: PC, prevention & control
CN 0 (Antibodies, Monoclonal); 0 (Antineoplastic Agents); 0 (****Bacterial*** Proteins); 0 (Drug Carriers); 0 (Membrane Lipids); EC 3.5.1.1 (Asparaginase)

L7 ANSWER 28 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
AN 1999:557792 CAPLUS <>LOGINID::20091202>>
DN 131:355959
TI ***bacterial*** ***ghosts*** as drug carrier and targeting vehicles
AU Huter, V.; Szostak, M. P.; Gampfer, J.; Prethaler, S.; Wanner, G.; Gabor, F.; Lubitz, W.
CS Institute of Microbiology and Genetics, University of Vienna, Vienna, A-1030, Austria
SO Journal of Controlled Release (1999), 61(1-2), 51-63
CODEN: JCREEC; ISSN: 0168-3659
PB Elsevier Science Ireland Ltd.
DT Journal
LA English
AB A novel system for the packaging of drugs as well as vaccines is presented. ***bacterial*** ***ghosts*** are intact, non-denatured ***bacterial*** envelopes that are created by lysis of ***bacteria*** through the expression of cloned phage PhiX174 gene E. Inhibition of induced E-mediated lysis by MgSO₄, harvesting of cells by centrifugation, and resuspension in low-ionic-strength buffers leads to rapid, violent lysis and results in empty ***bacterial*** envelopes with large (approx. 1 .mu.m in diam.) openings. The construction of plasmid pAV1, which encodes a streptavidin fusion protein with an N-terminal membrane anchor sequence, allows the loading of the inner side of the cytoplasmic membrane with streptavidin. The functionality and efficacy of binding of even large biotinylated compds. in such streptavidin ***ghosts*** (SA- ***ghosts***) was assessed using the enzyme alk. phosphatase. The successful binding of biotinylated fluorescent dextran, as well as fluorescent DNA complexed with biotinylated polylysine, was demonstrated microscopically. The display by ***bacterial*** ***ghosts*** of morphol. and antigenic surface structures of their living counterparts permits their attachment to target tissues such as the mucosal surfaces of the gastrointestinal and respiratory tract, and their uptake by phagocytes and M cells. In consequence, SA- ***ghosts*** are proposed as drug carriers for site-specific drug delivery.
OSC.G 27 THERE ARE 27 CAPLUS RECORDS THAT CITE THIS RECORD (27 CITINGS)
RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

- TI ***Bacterial*** ***ghosts*** as drug carrier and targeting vehicles
- AB A novel system for the packaging of drugs as well as vaccines is presented. ***Bacterial*** ***ghosts*** are intact, non-denatured ***bacterial*** envelopes that are created by lysis of ***bacteria*** through the expression of cloned phage PhiX174 gene E. Inhibition of induced E-mediated lysis by MgSO₄, harvesting of cells by centrifugation, and resuspension in low-ionic-strength buffers leads to rapid, violent lysis and results in empty ***bacterial*** envelopes with large (approx. 1 .mu.m in diam.) openings. The construction of plasmid pAV1, which encodes a streptavidin fusion protein. . . of the cytoplasmic membrane with streptavidin. The functionality and efficacy of binding of even large biotinylated compds. in such streptavidin ***ghosts*** (SA- ***ghosts***) was assessed using the enzyme alk. phosphatase. The successful binding of biotinylated fluorescent dextran, as well as fluorescent DNA complexed with biotinylated polylysine, was demonstrated microscopically. The display by ***bacterial*** ***ghosts*** of morphol. and antigenic surface structures of their living counterparts permits their attachment to target tissues such as the mucosal surfaces of the gastrointestinal and respiratory tract, and their uptake by phagocytes and M cells. In consequence, SA- ***ghosts*** are proposed as drug carriers for site-specific drug delivery.
- ST ***bacterial*** ***ghost*** drug delivery vehicle targeting
- IT ***Bacteria*** (Eubacteria)
- Drug targeting
- Genetic vectors
- Phagocytosis
 (***bacterial*** ***ghosts*** as drug carrier and targeting vehicles)
- IT Drug delivery systems
 (***bacterial*** ***ghosts*** ; ***bacterial***
 ghosts as drug carrier and targeting vehicles)
- IT Cell membrane
 (streptavidin-modified; ***bacterial*** ***ghosts*** as drug carrier and targeting vehicles)
- IT Biological transport
 (uptake, ***receptor*** -mediated; ***bacterial***
 ghosts as drug carrier and targeting vehicles)
- IT 9013-20-1D, Streptavidin, membrane conjugates
RL: BPR (Biological process); BSU (Biological study, unclassified); RCT (Reactant); BIOL (Biological study); PROC (Process); RACT (Reactant or reagent)
 (***bacterial*** ***ghosts*** as drug carrier and targeting vehicles)
- L7 ANSWER 29 OF 68 SCISEARCH COPYRIGHT (c) 2009 The Thomson Corporation on
STN
- AN 1998:503225 SCISEARCH <>LOGINID::20091202>>
- GA The Genuine Article (R) Number: ZX307
- TI ***Bacterial*** cell envelopes (***ghosts***) and LPS but not
 bacterial S-layers induce synthesis of immune-mediators in mouse
macrophages involving CD14
- AU Haslberger A G (Reprint)
- CS Univ Vienna, Bioctr, Inst Microbiol & Genet, Dr Bohrgasse 9, A-1030
Vienna, Austria (Reprint)
- AU Mader H J; Schmalnauer M; Kohl G; Szostak M P; Messner P; Sleytr U B;

Wanner G; Furst-Ladani S; Lubitz W
CS Univ Vienna, Bioctr, Inst Microbiol & Genet, A-1030 Vienna, Austria; Agr Univ Vienna, Zentrum Ultrastrukturforsch, A-1180 Vienna, Austria; Agr Univ Vienna, Ludwig Boltzmann Inst Mol Nanotechnol, A-1180 Vienna, Austria; Univ Munich, Inst Bot, D-8000 Munich, Germany; Lorenz Bohler Krankenhaus, Ludwig Boltzmann Inst Expt & Klin Traumatol, Vienna, Austria
CYA Austria; Germany
SO JOURNAL OF ENDOTOXIN RESEARCH, (DEC 1997) Vol. 4, No. 6, pp. 431-441.
ISSN: 0968-0519.
PB W S MANEY & SONS LTD, HUDSON RD, LEEDS LS9 7DL, ENGLAND.
DT Article; Journal
LA English
REC Reference Count: 46
ED Entered STN: 1998
Last Updated on STN: 1998
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The synthesis of inflammatory mediators in human macrophages/monocytes seen after stimulation with lipopolysaccharide (LPS) involves the binding of CD14 to LPS complexed to lipopolysaccharide binding protein (LBP). The binding mechanisms of different LPS domains to LBP and CD14, as well as the interaction of the entire ***bacterial*** cell wall and its components with CD14 and LBP, are poorly understood. We, therefore, studied the effects of anti-mouse CD14 antibodies on the synthesis of TNF alpha and PGE(2) in RAW 264.7 mouse macrophages stimulated by ***bacterial*** cell envelopes (***ghosts***) of Escherichia coli O26:B6 and Salmonella typhimurium C5, LPS, lipid A, and crystalline ***bacterial*** cell surface layer (S-layer) preparations. ***Ghosts*** and S-layers, with distinct activities on the immune-system, are presently under investigation for their use as vaccines. Whereas LPS and E. coli ***ghosts*** exhibited a strong endotoxic activity in the Limulus amoebocyte lysate assay, the endotoxic activity of S-layer preparations was several orders of magnitude lower. LPS, ***ghosts***, and ***bacterial*** S-layers all induced TNF alpha and PGE(2) synthesis as well as the accumulation of TNF alpha mRNA. Pre-incubation with anti-mouse CD14 antibodies resulted in a dose-dependent inhibition of TNF alpha and PGE(2) synthesis after stimulation by LPS, lipid A (30-50%) and ***ghosts*** (40-70%). The ***bacterial*** S-layer-induced mediator synthesis remained unchanged following the addition of anti-mouse CD14 antibodies. Reproducible differences could be observed for the inhibition of TNF alpha induced by LPS of different species by anti-CD14. Adding fetal calf serum (FCS) strongly enhanced the release of cell mediators stimulated by low doses of LPS and ***bacterial*** ***ghosts***. These effects of the FCS may be due to the presence of LBP in the FCS. The results show that CD14 is highly relevant for the activation of mouse macrophages by ***bacterial*** cells, LPS, and lipid A. Specially defined ***bacterial*** cell wall constituents such as ***bacterial*** S-layers might act through other activation pathways.
TI ***Bacterial*** cell envelopes (***ghosts***) and LPS but not ***bacterial*** S-layers induce synthesis of immune-mediators in mouse macrophages involving CD14
AB . . . (LBP). The binding mechanisms of different LPS domains to LBP and CD14, as well as the interaction of the entire ***bacterial*** cell wall and its components with CD14 and LBP, are poorly understood. We, therefore, studied the effects of anti-mouse CD14 antibodies on the synthesis of TNF alpha and PGE(2) in RAW 264.7 mouse macrophages stimulated by ***bacterial*** cell envelopes (***ghosts***) of

Escherichia coli O26:B6 and Salmonella typhimurium C5, LPS, lipid A, and crystalline ***bacterial*** cell surface layer (S-layer) preparations. ***Ghosts*** and S-layers, with distinct activities on the immune-system, are presently under investigation for their use as vaccines. Whereas LPS and E. coli ***ghosts*** exhibited a strong endotoxic activity in the Limulus amoebocyte lysate assay, the endotoxic activity of S-layer preparations was several orders of magnitude lower. LPS, ***ghosts***, and ***bacterial*** S-layers all induced TNF alpha and PGE(2) synthesis as well as the accumulation of TNF alpha mRNA. Pre-incubation with anti-mouse . . . antibodies resulted in a dose-dependent inhibition of TNF alpha and PGE(2) synthesis after stimulation by LPS, lipid A (30-50%) and ***ghosts*** (40-70%). The ***bacterial*** S-layer-induced mediator synthesis remained unchanged following the addition of anti-mouse CD14 antibodies. Reproducible differences could be observed for the inhibition. . . anti-CD14. Adding fetal calf serum (FCS) strongly enhanced the release of cell mediators stimulated by low doses of LPS and ***bacterial*** ***ghosts***. These effects of the FCS may be due to the presence of LBP in the FCS, the results show that CD14 is highly relevant for the activation of mouse macrophages by ***bacterial*** cells, LPS, and lipid A. Specially defined ***bacterial*** cell wall constituents such as ***bacterial*** S-layers might act through other activation pathways.

STP KeyWords Plus (R): TUMOR-NECROSIS-FACTOR; FACTOR-ALPHA GENE; BINDING-PROTEIN; TYROSINE PHOSPHORYLATION; LIPOPOLYSACCHARIDE LPS; MURINE MACROPHAGES; ESCHERICHIA-COLI; SURFACE-LAYERS; KAPPA-B; ***RECEPTOR***

L7 ANSWER 30 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN DUPLICATE 5
AN 1996:262954 BIOSIS <>LOGINID::20091202>>
DN PREV199698819083
TI Macrophage dysfunction following the phagocytosis of IgG-coated erythrocytes: Production of lipid peroxidation products.
AU Loegering, Daniel J. [Reprint author]; Raley, Michael J.; Reho, Thomas A.; Eaton, John W.
CS Dep. Physiol. Cell Biol., A-134, Albany Med. Coll., 47 New Scotland Ave., Albany, NY 12208-3479, USA
SO Journal of Leukocyte Biology, (1996) Vol. 59, No. 3, pp. 357-362.
CODEN: JLBIE7. ISSN: 0741-5400.
DT Article
LA English
ED Entered STN: 10 Jun 1996
Last Updated on STN: 10 Jun 1996
AB The phagocytosis of erythrocytes may contribute to the increased susceptibility to life-threatening infections in patients with burn injury, sickle cell anemia, and malaria. The phagocytosis of immunoglobulin G-coated erythrocytes (EIGG) is followed by a transient depression of several macrophage functions including phagocytosis, respiratory burst capacity, and killing of ***bacteria***. The present study suggests the possibility that after erythrophagocytosis hemoglobin-derived iron conspires with reactive oxygen products of the macrophage respiratory burst to cause oxidant damage to the phagocyte. Challenge of elicited peritoneal macrophages with EIGG phagocytosis was followed by an increase in lipid peroxidation as assessed by thiobarbituric acid-reactive substances (TBARS). Doses of EIGG associated with increased TBARS also caused a depression of Fc ***receptor***-mediated phagocytosis and phorbol myristate acetate (PMA)-stimulated

hydrogen peroxide production. Time course experiments demonstrated that the increase in TBARS coincided with the depression of macrophage function. There was no increase in TBARS following the phagocytosis of IgG-coated erythrocyte ***ghosts***, suggesting that hemoglobin iron is involved in the generation of TBARS. The phagocytosis of erythrocyte ***ghosts*** did not depress macrophage function. Since complement ***receptor*** -mediated phagocytosis does not stimulate the respiratory burst, the role of the respiratory burst in causing lipid peroxidation was assessed using the phagocytosis of complement-coated erythrocytes. Phagocytic challenge with complement-coated erythrocytes caused neither an increase in TBARS nor a depression of macrophage function. However, there was an increase in TBARS when the respiratory burst was stimulated with PMA following complement ***receptor*** -mediated phagocytosis of erythrocytes. These results suggest that hemoglobin iron and phagocyte-generated oxidants collaborate to cause the depression of macrophage function following EIgG phagocytosis.

- AB. . . erythrocytes (EIgG) is followed by a transient depression of several macrophage functions including phagocytosis, respiratory burst capacity, and killing of ***bacteria***. The present study suggests the possibility that after erytrophagocytosis hemoglobin-derived iron conspires with reactive oxygen products of the macrophage respiratory. . . as assessed by thiobarbituric acid-reactive substances (TBARS). Doses of EIgG associated with increased TBARS also caused a depression of Fc ***receptor*** -mediated phagocytosis and phorbol myristate acetate (PMA)-stimulated hydrogen peroxide production. Time course experiments demonstrated that the increase in TBARS coincided with the depression of macrophage function. There was no increase in TBARS following the phagocytosis of IgG-coated erythrocyte ***ghosts***, suggesting that hemoglobin iron is involved in the generation of TBARS. The phagocytosis of erythrocyte ***ghosts*** did not depress macrophage function. Since complement ***receptor*** -mediated phagocytosis does not stimulate the respiratory burst, the role of the respiratory burst in causing lipid peroxidation was assessed using. . . of macrophage function. However, there was an increase in TBARS when the respiratory burst was stimulated with PMA following complement ***receptor*** -mediated phagocytosis of erythrocytes. These results suggest that hemoglobin iron and phagocyte-generated oxidants collaborate to cause the depression of macrophage function. . .

- L7 ANSWER 31 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN DUPLICATE 6
AN 1994:227425 BIOSIS <>LOGINID::20091202>>
DN PREV199497240425
TI Pore-formation by Escherichia coli hemolysin (HlyA) and other members of the RTX toxins family.
AU Menestrina, Gianfranco [Reprint author]; Moser, Claudio; Pellet, Shahaireen; Welch, Rodney
CS CNR Centro di Fisica degli Stati Aggregati, via Sommarive 14, I-38050 Povo, Trento, Italy
SO Toxicology, (1994) Vol. 87, No. 1-3, pp. 249-267.
CODEN: TXCYAC. ISSN: 0300-483X.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 24 May 1994
Last Updated on STN: 24 May 1994
AB Escherichia coli hemolysin (HlyA) is a major cause of E. coli virulence.

It lyses erythrocytes by a colloid osmotic shock due to the formation of hydrophilic pores in the cell wall. The size of these channels can be estimated using osmotic protectant of increasing dimensions. To show that the formation of pores does not depend critically on the osmotic swelling we prepared resealed human erythrocyte ***ghosts*** loaded with a fluorescent marker. When attacked by HlyA the internal marker was released, indicating the formation of toxin channels so large as to let it through. The channels can be directly demonstrated also in purely lipidic model systems such as planar membranes and unilamellar vesicles, which lack any putative protein ***receptor***. HlyA has been recognised as a member of a large family of exotoxins elaborated by Gram-negative organisms including Proteus, Bordetella, Morganella, Pasteurella and Actinobacillus. These toxins have quite different target cell specificity and in many cases are leukocidal. When tried on planar membranes however, even specific leukotoxins open channels not dissimilar from those formed by HlyA, suggesting this might be a common step in their action. Comparison of the hydrophobic properties of six members of the toxin family indicates the presence of a conserved cluster of ten contiguous amphipathic helixes, located in the N-terminal half of the molecule, which might be involved in channel formation.

- AB. . . To show that the formation of pores does not depend critically on the osmotic swelling we prepared resealed human erythrocyte ***ghosts*** loaded with a fluorescent marker. When attacked by HlyA the internal marker was released, indicating the formation of toxin channels. . . directly demonstrated also in purely lipidic model systems such as planar membranes and unilamellar vesicles, which lack any putative protein ***receptor***. HlyA has been recognised as a member of a large family of exotoxins elaborated by Gram-negative organisms including Proteus, Bordetella,. . .

ORGN Classifier

Enterobacteriaceae 06702

Super Taxa

Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria ; Microorganisms

Organism Name

Escherichia coli

Taxa Notes

Bacteria , Eubacteria, Microorganisms

- L7 ANSWER 32 OF 68 SCISEARCH COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 1993:12050 SCISEARCH <>LOGINID:20091202>>
GA The Genuine Article (R) Number: KE757
TI DEPRESSION OF MACROPHAGE RESPIRATORY BURST CAPACITY AND ARACHIDONIC-ACID RELEASE AFTER FC ***RECEPTOR*** -MEDIATED PHAGOCYTOSIS
AU SCHWACHA M G (Reprint); GUDEWICZ P W; SNYDER J A; LOEGERING D J
CS UNION UNIV, DEPT PHYSIOL & CELL BIOL, ALBANY, NY 12208
CYA USA
SO JOURNAL OF IMMUNOLOGY, (1 JAN 1993) Vol. 150, No. 1, pp. 236-245.
ISSN: 0022-1767.
PB AMER ASSOC IMMUNOLOGISTS, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 48
ED Entered STN: 1994
Last Updated on STN: 1994

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The phagocytosis of IgG-coated erythrocytes (EIgG) by macrophages results in a subsequent depression of macrophage phagocytic function, respiratory burst capacity, and ***bactericidal*** activity. Our study was carried out to determine the importance of impaired arachidonic acid release in the depression of the respiratory burst after EIgG phagocytosis. The depression of triggered H2O2 production after EIgG phagocytosis was not due to cyclooxygenase products because indomethacin or aspirin did not modify the depression. Further studies revealed that the depression of triggered H2O2 production after EIgG phagocytosis was associated with a depression in the ability of macrophages to release arachidonic acid in response to PMA, zymosan, or calcium ionophore. The addition of exogenous arachidonic acid partially prevented the depression of triggered H2O2 production after EIgG phagocytosis. Unlike phagocytosis mediated by FcR, complement ***receptor*** -mediated phagocytosis did not alter H2O2 production or arachidonic acid release. Ligation of FcR was not sufficient to depress triggered H2O2 production and arachidonic acid release because these functions were not depressed when phagocytosis was inhibited with cytochalasin D. Thus, it was found that the depression of triggered H2O2 production by macrophages after FcR-mediated phagocytosis was associated with impaired release of arachidonic acid and that H2O2 production could be partially restored by the addition of arachidonic acid. These results suggest that the impairment of arachidonic acid release after FcR-mediated phagocytosis contributes to the depression of macrophage respiratory burst capacity after FcR-mediated phagocytosis.

TI DEPRESSION OF MACROPHAGE RESPIRATORY BURST CAPACITY AND ARACHIDONIC-ACID RELEASE AFTER FC ***RECEPTOR*** -MEDIATED PHAGOCYTOSIS

AB . . . phagocytosis of IgG-coated erythrocytes (EIgG) by macrophages results in a subsequent depression of macrophage phagocytic function, respiratory burst capacity, and ***bactericidal*** activity. Our study was carried out to determine the importance of impaired arachidonic acid release in the depression of the . . . exogenous arachidonic acid partially prevented the depression of triggered H2O2 production after EIgG phagocytosis. Unlike phagocytosis mediated by FcR, complement ***receptor*** -mediated phagocytosis did not alter H2O2 production or arachidonic acid release. Ligation of FcR was not sufficient to depress triggered H2O2. . .

STP KeyWords Plus (R): HYDROGEN-PEROXIDE PRODUCTION; NEUTROPHIL NADPH-OXIDASE; UNSATURATED FATTY-ACIDS; SUPEROXIDE GENERATION; ERYTHROCYTE- ***GHOSTS*** ; PROTEIN-SYNTHESIS; HOST DEFENSE; ACTIVATION; OXYGEN; INHIBITORS

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AN 1993:165142 BIOSIS <>LOGINID::20091202>

DN PREV199395086192

TI Fc-epsilon-RI-mediated hydrolysis of phosphoinositides in permeable membrane vesicles.

AU Kuhn, Donald E.; Dreskin, Stephen C. [Reprint author]

CS B164, Univ. Colo. Sch. Med., 4200 East Ninth Ave., Denver, CO 80262, USA

SO Journal of Immunological Methods, (1993) Vol. 157, No. 1-2, pp. 81-89.

CODEN: JIMMBG. ISSN: 0022-1759.

DT Article

LA English

ED Entered STN: 31 Mar 1993

Last Updated on STN: 1 Apr 1993

AB We have used hypotonic lysis of cytoplasm derived from rat basophilic

leukemia (RBL) cell to prepare organelle and cytoplasm-depleted membrane vesicles called 'RBL cell ***ghosts***' (Dreskin and Metzger, 1991). Unlike other membrane preparations, the RBL ***ghosts*** hydrolyze phosphoinositides (PIs) in response to aggregation of the high affinity ***receptor*** for IgE (Fc-epsilon-RI) and have proven useful for studies of the molecular events involved in transduction of this signal. A significant limitation of these preparations is that they are sealed. Thus, to incorporate membrane-impermeant molecules (such as ATP) into the intravesicular space of the ***ghosts***, they must be added as the ***ghosts*** are formed. We have now overcome this problem by permeabilizing the ***ghosts*** with alpha-toxin from *S. aureus* and find that, following permeabilization, ***ghosts*** activated via Fc-epsilon-RI, hydrolyze PIs for a longer time than do non-permeabilized ***ghosts***. As in the intact ***ghosts***, this response is absolutely dependent upon ATP and is enhanced by the addition of either phosphoenolpyruvate (PEP) or creatine phosphate (CP). This report demonstrates that we can now manipulate the intravesicular environment of the RBL ***ghosts*** and extends the utility of these preparations as a model system for the study of signal transduction following activation via Fc-epsilon-RI.

AB. . . lysis of cytoplasm derived from rat basophilic leukemia (RBL) cell to prepare organelle and cytoplasm-depleted membrane vesicles called 'RBL cell ***ghosts***' (Dreskin and Metzger, 1991). Unlike other membrane preparations, the RBL ***ghosts*** hydrolyze phosphoinositides (PIs) in response to aggregation of the high affinity ***receptor*** for IgE (Fc-epsilon-RI) and have proven useful for studies of the molecular events involved in transduction of this signal. A . . . preparations is that they are sealed. Thus, to incorporate membrane-impermeant molecules (such as ATP) into the intravesicular space of the ***ghosts***, they must be added as the ***ghosts*** are formed. We have now overcome this problem by permeabilizing the ***ghosts*** with alpha-toxin from *S. aureus* and find that, following permeabilization, ***ghosts*** activated via Fc-epsilon-RI, hydrolyze PIs for a longer time than do non-permeabilized ***ghosts***. As in the intact ***ghosts***, this response is absolutely dependent upon ATP and is enhanced by the addition of either phosphoenolpyruvate (PEP) or creatine phosphate (CP). This report demonstrates that we can now manipulate the intravesicular environment of the RBL ***ghosts*** and extends the utility of these preparations as a model system for the study of signal transduction following activation via . . .

IT Miscellaneous Descriptors

ALPHA TOXIN PERMEABILIZING AGENT; ATP DEPENDENT; CREATINE PHOSPHATE;
HIGH AFFINITY IMMUNOGLOBULIN E ***RECEPTOR*** ; HIGH ENERGY
PHOSPHATE COMPOUND ENHANCEMENT; METHOD; PHOSPHOENOLPYRUVATE; RAT
BASOPHILIC LEUKEMIA CELL ***GHOSTS*** ; SIGNAL TRANSDUCTION

ORGN Classifier

Micrococcaceae 07702

Super Taxa

Gram-Positive Cocci; Eubacteria; ***Bacteria*** ; Microorganisms

Organism Name

Staphylococcus aureus

Taxa Notes

Bacteria , Eubacteria, Microorganisms

ORGN Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Organism Name
Muridae
Taxa Notes
Animals, Chordates, . . .

L7 ANSWER 34 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN DUPLICATE 7

AN 1992:456099 BIOSIS <>LOGINID::20091202>>

DN PREV199294097499; BA94:97499

TI SURVEY OF A ***RECEPTOR*** PROTEIN IN HUMAN ERYTHROCYTES FOR
HEMAGGLUTININ OF PORPHYROMONAS-GINGIVALIS.

AU HAYASHI H [Reprint author]; NAGATA A; HINODE D; SATO M; NAKAMURA R

CS DEP OF PREVENTIVE DENTISTRY, UNIV OF TOKUSHIMA SCH OF DENT, 18-15,
KURAMOTOCHO-3-CHROME, TOKUSHIMA CITY 770, JAPAN

SO Oral Microbiology and Immunology, (1992) Vol. 7, No. 4, pp. 204-211.
CODEN: OMIMEE. ISSN: 0902-0055.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 7 Oct 1992
Last Updated on STN: 7 Oct 1992

AB The purpose of this study is to survey a ***receptor*** protein in
human erythrocyte membrane for the hemagglutinin (HA) of Porphyromonas
gingivalis. Human erythrocytes were modified by either chymotrypsin or P.
gingivalis HA along with the disappearance of their hemagglutinating
ability and the removal of the band 3 protein. By preparative
electrophoresis, this protein was isolated and purified from human
erythrocytes. The purified protein showed strong inhibitory activity for
hemagglutination and the binding to P. gingivalis cells, whose binding
sites were calculated to be approximately 9000, suggesting its binding to
the active site of HA. Hemagglutinin purified from P. gingivalis by
affinity absorption to sheep erythrocyte ***ghosts*** possessed strong
trypsin-like activity, and both the HA and the enzyme activities were
inhibited by arginine. Specific modification of arginyl residues in human
erythrocytes by phenylglyoxal diminished the hemagglutinating ability.
From the similarity of the inhibition profile and possible active sites
between HA and the trypsin-like protease, it is suggested that
hemagglutination may occur as a result of the primary reaction of the
enzyme (protease) and the substrate. These results suggest that band 3
may be a key protein in human erythrocyte membrane for HA from P.
gingivalis and its binding sites may be arginyl residues of the protein.

TI SURVEY OF A ***RECEPTOR*** PROTEIN IN HUMAN ERYTHROCYTES FOR
HEMAGGLUTININ OF PORPHYROMONAS-GINGIVALIS.

AB The purpose of this study is to survey a ***receptor*** protein in
human erythrocyte membrane for the hemagglutinin (HA) of Porphyromonas
gingivalis. Human erythrocytes were modified by either chymotrypsin or
. . suggesting its binding to the active site of HA. Hemagglutinin
purified from P. gingivalis by affinity absorption to sheep erythrocyte
ghosts possessed strong trypsin-like activity, and both the HA
and
the enzyme activities were inhibited by arginine. Specific modification
of arginyl. . .

ORGN Classifier
Bacteroidaceae 06901

Super Taxa
Anaerobic Gram-Negative Rods; Eubacteria; ***Bacteria*** ;
Microorganisms

Taxa Notes
 Bacteria , Eubacteria, Microorganisms

ORGN Classifier
 Hominidae 86215
 Super Taxa
 Primates; Mammalia; Vertebrata; Chordata; Animalia
 Taxa Notes
 Animals, Chordates, Humans, Mammals, Primates,.. . .

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STN DUPPLICATE 8

AN 1992:236269 BIOSIS <>LOGINID::20091202>>

DN PREV199293124294; BA93:124294

TI SOLUBILIZATION OF THE BINDING PROTEIN FROM EHRLICH ASCITES CELLS AND
ERYTHROCYTES TO PSEUDOMONAS-AERUGINOSA CYTOTOXIN.

AU JUNGBLUT R [Reprint author]; GRIMMIG M; LEIDOLF R; LUTZ F

CS INST PHARMAKOLOGIE TOXIKOLOGIE, JUSTUS-LIEBIG-UNIVERSITAET GIESSEN,
FRANKFURTER STR 107, W-6300 GIESSEN, GERMANY

SO Biological Chemistry Hoppe-Seyler, (1992) Vol. 373, No. 2, pp. 93-100.
CODEN: BCHSEI. ISSN: 0177-3593.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 10 May 1992
Last Updated on STN: 10 May 1992

AB The binding protein for pore-forming *Pseudomonas aeruginosa* cytotoxin was solubilized from Ehrlich ascites cell plasma membranes and rabbit and bovine erythrocyte ***ghosts*** using nonionic and zwittergent detergents. Analysis of solubilized plasma membranes from Ehrlich cells by a ***ligand*** -blot technique after separation by SDS-PAGE/electrophoretic transfer to nitrocellulose or affinity chromatography showed a protein of 70 kDa molecular mass, which binds to cytotoxin. The binding protein solubilized from rabbit erythrocyte ***ghosts*** showed a molecular mass of 50 kDa and that from bovine ***ghosts*** 55 kDa according to the former test. The binding proteins could be characterized as acidic. They contain a glycan moiety which is, however, not involved in the interaction of cytotoxin with the binding site.

AB. . . binding protein for pore-forming *Pseudomonas aeruginosa* cytotoxin was solubilized from Ehrlich ascites cell plasma membranes and rabbit and bovine erythrocyte ***ghosts*** using nonionic and zwittergent detergents. Analysis of solubilized plasma membranes from Ehrlich cells by a ***ligand*** -blot technique after separation by SDS-PAGE/electrophoretic transfer to nitrocellulose or affinity chromatography showed a protein of 70 kDa molecular mass, which binds to cytotoxin. The binding protein solubilized from rabbit erythrocyte ***ghosts*** showed a molecular mass of 50 kDa and that from bovine ***ghosts*** 55 kDa according to the former test. The binding proteins could be characterized as acidic. They contain a glycan moiety. . .

ORGN Classifier
 Pseudomonadaceae 06508
 Super Taxa
 Gram-Negative Aerobic Rods and Coccii; Eubacteria; ***Bacteria*** ;
 Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

ORGN Classifier

Bovidae 85715
Super Taxa
Artiodactyla; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Artiodactyls, Chordates, Mammals, Nonhuman. . .

L7 ANSWER 36 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
AN 1992:1761 CAPLUS <>LOGINID:20091202>>
DN 116:1761
OREF 116:363a,366a
TI Membrane-anchoring of heterologous proteins in recombinant hosts for use
as antigens
IN Lubitz, Werner; Szostak, Michael P.
PA Boehringer Mannheim G.m.b.H., Germany
SO PCT Int. Appl., 46 pp.
CODEN: PIXXD2
DT Patent
LA German
FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------|---|------|----------|-----------------|----------|
| PI | WO 9113155 | A1 | 19910905 | WO 1991-EP308 | 19910219 |
| | W: AU, FI, HU, JP, SU, US | | | | |
| | RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE | | | | |
| | DE 4005874 | A1 | 19911107 | DE 1990-4005874 | 19900224 |
| | AU 9172373 | A | 19910918 | AU 1991-72373 | 19910219 |
| | EP 516655 | A1 | 19921209 | EP 1991-903789 | 19910219 |
| | EP 516655 | B1 | 19940504 | | |
| | R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE | | | | |
| | JP 05503014 | T | 19930527 | JP 1991-503980 | 19910219 |
| | JP 3238396 | B2 | 20011210 | | |
| | AT 105335 | T | 19940515 | AT 1991-903789 | 19910219 |
| | US 5470573 | A | 19951128 | US 1992-924028 | 19920930 |
| PRAI | DE 1990-4005874 | A | 19900224 | | |
| | EP 1991-903789 | A | 19910219 | | |
| | WO 1991-EP308 | A | 19910219 | | |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Antigenic proteins are prep'd. with a Gram-neg. ***bacteria*** contg. a
gene encoding a lytic protein by expression of a chimeric gene for a
fusion protein of a membrane-anchoring domain and the antigen. Plasmid
pAV5 encoding a streptavidin-phage MS2 protein L fusion protein and a
plasmid contg. the protein E gene of phage *phi*X174 under control of the
temp. sensitive lambda. repressor-lambda. promoter/operator system were
prep'd. Escherichia coli was transformed with these plasmids, cultured to
permit cell growth and fusion protein synthesis, then temp.-shifted to
cause protein E prodn. and cell lysis. The ***bacterial***
ghosts prep'd. were incubated with a hepatitis B core antigen-
biotin conjugate to prep. an immunogen.

OSC.G 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)
RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB Antigenic proteins are prep'd. with a Gram-neg. ***bacteria*** contg. a
gene encoding a lytic protein by expression of a chimeric gene for a
fusion protein of a membrane-anchoring. . . cultured to permit cell
growth and fusion protein synthesis, then temp.-shifted to cause protein E
prodn. and cell lysis. The ***bacterial*** ***ghosts*** prep'd.
were incubated with a hepatitis B core antigen- ***biotin*** conjugate

ST to prep. an immunogen.

ST antigen membrane anchor fusion Escherichia; lytic protein
 bacterial ***ghost*** immunogen; vaccine recombinant
 bacteria ***ghost***

IT Vaccines
 (***bacterial*** ***ghosts*** contg. membrane-assocd.
 recombinant antigens for, prepn. of)

IT Avidins

RL: PREP (Preparation)
 (fusion products with membrane-anchoring domains, recombinant manuf. in
 Escherichia coli of, prepn. of cell ***ghosts*** for vaccines of,
 bacteriophage lytic functions in)

IT Antigens

RL: PREP (Preparation)
 (fusion proteins with membrane-anchoring domains of, Gram-neg.
 bacterial ***ghosts*** contg., prepn. of,
 bacteriophage lytic functions in, vaccines in relation to)

IT Escherichia coli
 (***ghosts*** of, antigens anchored to membranes of,
 bacteriophage lytic functions in, vaccines in relation to)

IT Virus, ***bacterial***
 .lytic functions of, in prepn. Gram-neg. ***bacterial***
 ghosts contg. antigen-membrane-anchoring domain fusion
 proteins, vaccines in relation to)

IT Proteins, biological studies

RL: PREP (Preparation)
 .lytic, of ***bacteriophage*** , in prepn. Gram-neg.
 bacterial ***ghosts*** contg. of antigen-membrane-anchoring
 domain fusion proteins, vaccines in relation to)

IT Mammal
 (vaccines for, antigens for, ***bacterial*** ***ghosts***
 contg. membrane-assocd. recombinant antigens as)

IT Proteins, specific or class

RL: PREP (Preparation)
 (E, of ***bacteriophage*** .phi.X174, in prepn. of Gram-neg.
 bacterial ***ghosts*** contg. antigen-membrane-anchoring
 domain fusion proteins, vaccines in relation to)

IT Proteins, specific or class

RL: PREP (Preparation)
 (L, of ***bacteriophage*** MS2, in prepn. of Gram-neg.
 bacterial ***ghosts*** contg. antigen-membrane-anchoring
 domain fusion proteins, vaccines in relation to)

IT Virus, ***bacterial***
 (MS2, protein L of, in prepn. Gram-neg. ***bacterial***
 ghosts contg. antigen-membrane-anchoring domain fusion
 proteins, vaccines in relation to)

IT Gene

RL: BIOL (Biological study)
 (chimeric, for fusion proteins of antigens and membrane-anchoring
 domains, expression in Gram-neg. ***bacteria*** of, vaccines in
 relation to)

IT Proteins, specific or class

RL: BIOL (Biological study)
 (fusion products, of antigens with membrane-anchoring domain, manuf. in
 Gram-neg. ***bacteria*** of, ***bacteriophage*** lytic
 functions in, vaccine manuf. in relation to)

IT Sialoglycoproteins
RL: PREP (Preparation)
(gp120env, fusion products, with ***bacteriophage*** proteins E or L, membrane anchoring in Escherichia coli of, prepn. of ***ghosts*** for vaccines in relation to)

IT Glycoproteins, specific or class
RL: PREP (Preparation)
(gp41env, fusion products, with ***bacteriophage*** proteins E or L, membrane anchoring in Escherichia coli of, prepn. of ***ghosts*** for vaccines in relation to)

IT ***Bacteria***
(gram-neg., membrane anchoring of heterologous proteins in, membrane-anchoring domains and ***bacteriophage*** lytic functions in, vaccines in relation to)

IT Antigens
RL: BIOL (Biological study)
(hepatitis B core, conjugate with ***biotin***, complex with Escherichia coli ***ghosts*** contg. membrane-bound streptavidin, as immunogen)

IT Virus, ***bacterial***
(phi X174, protein E of, in prepn. Gram-neg. ***bacterial*** ***ghosts*** contg. antigen-membrane-anchoring domain fusion proteins, vaccines in relation to)

IT 137925-62-3, Deoxyribonucleic acid (Escherichia coli clone pMC1403 gene lacZ plus 3'-flanking region fragment) 137925-65-6 137926-10-4, Deoxyribonucleic acid (Streptomyces avidinii clone pAV5 streptavidin gene plus 5'- and 3'-flanking region fragment)
RL: BIOL (Biological study)
(chimeric gene contg., for fusion protein of membrane-anchoring domain and antigenic determinant, expression in Escherichia coli of, ***bacteriophage*** lytic functions in)

IT 9013-20-1D, Streptavidin, fusion products with membrane-anchoring protein 9031-11-2D, .beta.-Galactosidase, fusion products with phage E or L proteins
RL: BIOL (Biological study)
(membrane-bound, recombinant manuf. in Escherichia coli of, prepn. of cell ***ghosts*** for vaccines of, ***bacteriophage*** lytic functions in)

L7 ANSWER 37 OF 68 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

AN 1992046723 EMBASE <>LOGIND::20091202>>

TI Comparison of hemagglutinating pili of Haemophilus influenzae type b with similar structures of nontypeable H. influenzae.

AU Gilsdorf, J.R. (correspondence); Chang, H.Y.; McCrea, K.W.; Bakalatz, L.O.

CS Department of Pediatrics, University of Michigan, Ann Arbor, MI 48109-0244, United States.

SO Infection and Immunity, (1991) Vol. 60, No. 2, pp. 374-379.
ISSN: 0019-9567 CODEN: INFIBR

CY United States

DT Journal; Article

FS 004 Microbiology: Bacteriology, Mycology, Parasitology and Virology

LA English

SL English

ED Entered STN: 20 Mar 1992
Last Updated on STN: 20 Mar 1992

AB Thirty-eight clinical isolates of nontypeable Haemophilus influenzae were

tested for the presence of hemagglutinating pili similar to those of *H. influenzae* type b (Hib) that mediate buccal epithelial cell adherence. Four endogenously hemagglutinating (HA+) strains were identified, and eight additional HA+ variants were obtained from HA- strains by erythrocyte enrichment. All 12 HA+ nontypeable *H. influenzae* isolates bound antisera directed against denatured pilins of Hib, but none bound antisera against assembled native pili of Hib. In erythrocyte- and buccal-cell-binding assays, HA+ nontypeable *H. influenzae* binding was reduced compared with HA+ Hib binding and was not significantly different from HA- nontypeable *H. influenzae* binding. Both HA- and HA+ nontypeable *H. influenzae* binding was increased over binding of HA- Hib. HA+ nontypeable *H. influenzae* strains agglutinated adult erythrocytes that possess the Anton antigen, which is thought to be the ***receptor*** for Hib pili, and did not agglutinate cord or Lu(a-b-) dominant erythrocytes, which lack the Anton antigen. Electron microscopy of HA- and HA+ variants of three nontypeable *H. influenzae* strains showed few or no surface appendages on the HA- organisms, but piluslike structures were seen on many organisms from two HA+ nontypeable *H. influenzae* strains and on a few organisms from one strain. Thus, nontypeable *H. influenzae* appears to possess structures that are immunologically similar to the pilins that make up the hemagglutinating pili of Hib. However, nontypeable *H. influenzae* appears to also possess mechanisms for erythrocyte and buccal cell adherence that are not directly correlated with the presence of a hemagglutinating pilus.

AB . . . Hib. HA+ nontypeable *H. influenzae* strains agglutinated adult erythrocytes that possess the Anton antigen, which is thought to be the ***receptor*** for Hib pili, and did not agglutinate cord or Lu(a-b-) dominant erythrocytes, which lack the Anton antigen. Electron microscopy of. . .

CT Medical Descriptors:

article

****bacterium adherence***

****bacterium identification***

****bacterium pilus***

cheek mucosa

comparative study

controlled study

electron microscopy

epithelium cell

erythrocyte ghost

*haemophilus influenzae type b

*hemagglutination

human

human cell

pasteurellaceae

phenotype

priority journal

*polyclonal antibody

*rabbit antiserum

L7 ANSWER 38 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

AN 1991:70460 BIOSIS <>LOGINID::20091202>>

DN PREV199191039120; BA91:39120

TI INHIBITION OF CHOLERA TOXIN BINDING TO MEMBRANE RECEPTORS BY PIG GASTRIC MUCIN-DERIVED GLYCOPEPTIDES DIFFERENTIAL EFFECT DEPENDING ON THE ABO BLOOD GROUP ANTIGENIC DETERMINANTS.

AU MONFERRAN C G [Reprint author]; ROTH G A; CUMAR F A
CS DEP DE QUIMICA BIOL, FAC DE CIENCIAS QUIMICAS, UNIV NACIONAL DE CORDOBA,
CIQUIBIC-CONICET, 5016 CORDOBA, ARGENTINA
SO Infection and Immunity, (1990) Vol. 58, No. 12, pp. 3966-3972.
CODEN: INFIBR. ISSN: 0019-9567.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 29 Jan 1991
Last Updated on STN: 30 Jan 1991
AB The capacity of pig gastric mucin-derived glycopeptides to interfere with the binding of cholera toxin (CT) to membrane receptors was studied. Two types of glycopeptide preparations with or without human blood group A antigenic activity were assayed for comparison in a system in which the target for the toxin was rat erythrocyte ***ghosts***. Blood group A-active glycopeptides (A+ glycopeptides) were more potent inhibitors for the toxin binding than those lacking group A activity (A- glycopeptides). The mean values of the 50% inhibitory dose revealed that the A+ glycopeptide preparations were 6.6-fold-more potent inhibitors than the A- ones ($P < 0.001$). The inhibitory capacity of the different A+ glycopeptide preparations was not directly proportional to the group A antigenic titer. The A+ glycopeptides showed a higher capacity than the A- glycopeptides to interact with the toxin as revealed by CT-glycopeptide complex formation, which could be detected by Sephadryl S-400 chromatography. This result suggests that glycopeptide inhibition of CT binding to the erythrocyte ***ghosts*** is mediated by a competition between the GM1 receptors and the glycopeptides for the toxin. The differential effect between both types of glycoconjugates was independent of the way of measuring the amount of glycopeptides used (dry weight, carbohydrate or protein content). The existence in the gastrointestinal tract of mucins not carrying or carrying different ABO blood group determinants, which could behave as more or less potent inhibitors of CT binding to membrane receptors, may help to explain the relationship between ABO blood groups and severity of cholera.
AB. . . A antigenic activity were assayed for comparison in a system in which the target for the toxin was rat erythrocyte ***ghosts***. Blood group A-active glycopeptides (A+ glycopeptides) were more potent inhibitors for the toxin binding than those lacking group A activity. . which could be detected by Sephadryl S-400 chromatography. This result suggests that glycopeptide inhibition of CT binding to the erythrocyte ***ghosts*** is mediated by a competition between the GM1 receptors and the glycopeptides for the toxin. The differential effect between both. .
IT Miscellaneous Descriptors
 ANTIGENIC TITER GLYCOCONJUGATE TYPES ***RECEPTOR*** COMPETITION
ORGN Classifier
 Vibrionaceae 06704
 Super Taxa
 Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
 Bacteria ; Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms
ORGN Classifier
 Suidae 85740
 Super Taxa
 Artiodactyla; Mammalia; Vertebrata; Chordata; Animalia
 Taxa Notes

Animals, Artiodactyls, Chordates, Mammals, Nonhuman. . .

L7 ANSWER 39 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN DUPLICATE 9
AN 1991:114458 BIOSIS <>LOGINID::20091202>>
DN PREV199191061848; BA91:61848
TI EFFECT OF PHAGOCYTOSIS OF ERYTHROCYTES AND ERYTHROCYTE ***GHOSTS*** ON
MACROPHAGE PHAGOCYTIC FUNCTION AND HYDROGEN PEROXIDE PRODUCTION.
AU COMMINS L M [Reprint author]; LOEGERING D J; GUDEWICZ P W
CS DEP PHYSIOLOGY CELL BIOL, ALBANY MED COLL, 47 NEW SCOTLAND AVENUE, ALBANY,
NEW YORK 12208, USA
SO Inflammation, (1990) Vol. 14, No. 6, pp. 705-716.
CODEN: INFLED4. ISSN: 0360-3997.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 27 Feb 1991
Last Updated on STN: 28 Feb 1991
AB Our previous studies have shown that an in vivo phagocytic challenge with
IgG-coated erythrocytes can depress Kupffer cell complement and Fc
receptor function, as well as decrease the survival rate
following
endotoxemia and ***bacteremia***. In an effort to better understand
the mechanism underlying these in vivo findings, the present study
evaluated the in vitro effects of a phagocytic challenge with either
IgG-coated erythrocytes (EIgG) or erythrocyte ***ghosts*** (GIgG) on
macrophage phagocytic and respiratory burst activity. Elicited rat
peritoneal macrophage (PM) monolayers were challenged with varying doses
of EIgG, then the noninternalized EIgG were lysed hypotonically and the
monolayers incubated for an additional hour prior to determining
phagocytic function and PMA-stimulated hydrogen peroxide production.
Challenge of PM with 1 .times. 106 EIgG per well had no effect, but
challenge with 1 .times. 107 or 1 .times. 108 EIgG per well caused a
dose-dependent depression of phagocytic function or hydrogen peroxide
production. GIgG were formed by hypotonically lysing EIgG bound to PM at
4.degree. C. The bound GIgG were phagocytized during a subsequent
incubation at 37.degree. C. Challenge with GIgG depressed phagocytic
function only with the highest challenge dose tested (1 .times. 108 per
well) and did not depress hydrogen peroxide production. The observation
that prior phagocytic challenge with EIgG depressed macrophage function to
a greater extent than challenge with GIgG supports our previous in vivo
observations. Furthermore, these studies suggest that the internalization
of erythrocyte contents, and not phagocytosis per se, plays an important
role in determining macrophage host defense function.
TI EFFECT OF PHAGOCYTOSIS OF ERYTHROCYTES AND ERYTHROCYTE ***GHOSTS*** ON
MACROPHAGE PHAGOCYTIC FUNCTION AND HYDROGEN PEROXIDE PRODUCTION.
AB . . . previous studies have shown that an in vivo phagocytic challenge
with IgG-coated erythrocytes can depress Kupffer cell complement and Fc
receptor function, as well as decrease the survival rate
following
endotoxemia and ***bacteremia***. In an effort to better understand
the mechanism underlying these in vivo findings, the present study
evaluated the in vitro effects of a phagocytic challenge with either
IgG-coated erythrocytes (EIgG) or erythrocyte ***ghosts*** (GIgG) on
macrophage phagocytic and respiratory burst activity. Elicited rat
peritoneal macrophage (PM) monolayers were challenged with varying doses
of. . .

L7 ANSWER 40 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

AN 1990:26846 BIOSIS <>LOGINID::20091202>>

DN PREV199089013812; BA89:13812

TI NEURAMINIDASE OF NONPATHOGENIC ARTHROBACTER-SP.

AU KOTLYAR T V [Reprint author]; SHATAEVA L K; ZAIKINA N A; CHERNOVA I A; ABRASHEV I R

CS LENINGR CHEM PHARM INST, LENINGRAD, USSR

SO Prikladnaya Biokhimiya i Mikrobiologiya, (1989) Vol. 25, No. 4, pp. 467-472.

CODEN: PBMIAK. ISSN: 0555-1099.

DT Article

FS BA

LA RUSSIAN

ED Entered STN: 19 Dec 1989
Last Updated on STN: 20 Dec 1989

AB Neuraminidase of the nonpathogenic microorganism Arthrobacter nicotianae was studied by gel chromatography and affinity chromatography on "Stropan"-an adsorbent containing erythrocyte ***ghosts*** incorporated in a porous polymeric matrix. The molecular weight of the enzyme was found to be 170 .+-. 20 kDa. The conditions of the neuraminidase adsorption on Stropan were optimized. The neuraminidase preparation obtained is comparable with commercial neuraminidase from noncholerae vibrios by the specific activity and the specific effect on receptors of human erythrocytes.

AB. . . Neuraminidase of the nonpathogenic microorganism Arthrobacter nicotianae was studied by gel chromatography and affinity chromatography on "Stropan"-an adsorbent containing erythrocyte ***ghosts*** incorporated in a porous polymeric matrix. The molecular weight of the enzyme was found to be 170 .+-. 20 kDa.. . .

IT Miscellaneous Descriptors
HUMAN ERYTHROCYTE ***RECEPTOR*** OPTIMIZATION AFFINITY CHROMATOGRAPHY BIOTECHNOLOGY INDUSTRY

ORGN Classifier
Irregular Nonsporing Gram-Positive Rods 08890
Super Taxa
Actinomycetes and Related Organisms; Eubacteria; ***Bacteria*** ;
Microorganisms
Taxa Notes
Bacteria , Eubacteria, Microorganisms

ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates,. . .

L7 ANSWER 41 OF 68 LIFESCI COPYRIGHT 2009 CSA on STN
AN 89:169 LIFESCI <>LOGINID::20091202>>

TI Novel lectins derived from ***bacterial*** pili.

AU Brinton, C.C., Jr.; Hanson, M.

CS Bactex, Inc., Pittsburgh, PA (USA)

PI US 4801690 1989

SO (1989) . US Cl. 530/396; Int. Cl. A61K 37/02, 39/108, 39/112, C12N 1/00..

DT Patent

FS A; W

LA English
 AB The authors describe a bactolectin derived from the pili of an organism selected from the group consisting of *E. coli*, and *Salmonella* species, said ***lectin*** being non-covalently bindable to the pilus rod protein of said pili and separable therefrom by the action of hot aqueous sodium dodecyl sulfate and possessing at least a single binding site for binding to mammalian erythrocyte ***ghosts***.
 TI Novel lectins derived from ***bacterial*** pili.
 AB . . . derived from the pili of an organism selected from the group consisting of *E. coli*, and *Salmonella* species, said ***lectin*** being non-covalently bindable to the pilus rod protein of said pili and separable therefrom by the action of hot aqueous sodium dodecyl sulfate and possessing at least a single binding site for binding to mammalian erythrocyte ***ghosts***.
 UT patents; lectins; *Escherichia coli*; *Salmonella*; pili; ***bacteria***
 L7 ANSWER 42 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 1988:183579 CAPLUS <>LOGINID::20091202>>
 DN 108:183579
 OREF 108:30088h,30089a
 TI Purifications of lectins containing mammalian erythrocyte binding sites from ***bacterial*** pili
 IN Brinton, Charles C., Jr.; Hanson, Mark
 PA Bactex, Inc., USA
 SO PCT Int. Appl., 27 pp.
 CODEN: PIXXD2
 DT Patent
 LA English
 FAN.CNT 1

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|------|----------|-----------------|----------|
| PI WO 8705910 | A1 | 19871008 | WO 1987-US617 | 19870320 |
| W: JP | | | | |
| RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE | | | | |
| US 4801690 | A | 19890131 | US 1986-842946 | 19860324 |
| EP 298991 | A1 | 19890118 | EP 1987-902927 | 19870320 |
| EP 298991 | B1 | 19920617 | | |
| R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE | | | | |
| JP 01501225 | T | 19890427 | JP 1987-502341 | 19870320 |
| JP 2778690 | B2 | 19980723 | | |
| AT 77392 | T | 19920715 | AT 1987-902927 | 19870320 |
| CA 1282323 | C | 19910402 | CA 1987-532748 | 19870323 |
| PRAI US 1986-842946 | A | 19860324 | | |
| US 1986-842947 | A | 19860324 | | |
| EP 1987-902927 | A | 19870320 | | |
| WO 1987-US617 | W | 19870320 | | |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB ***Lectin*** is isolated from pili of pilated ***bacteria***. The lectins are noncovalently bindable to the pilus rod protein of the pili and possess a single binding site for mammalian erythrocyte ***ghosts***. *Escherichia coli* Type I was suspended in aq. NaCl and blended. The product was centrifuged to remove cell debris. The supernatant was taken in buffer contg. SDS, boiled for 5 min, cooled, and aggregated pilin rods were sedimented by centrifugation. The supernatant contained 28-, 16.5-, and 14.5-kilodalton proteins, which were ptd. with aceton and sepd. by Sephadex gel column. Papain inactivation of pili was correlated with degrdn. of the 28-kilodalton protein on the pili. This

lectin was coupled to CNBr Sepharose for use as mannose-specific affinity resin.

OSC.G 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)
RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Purifications of lectins containing mammalian erythrocyte binding sites from ***bacterial*** pili

AB ***Lectin*** is isolated from pili of pilated ***bacteria*** . The lectins are noncovalently bindable to the pilus rod protein of the pili and possess a single binding site for mammalian erythrocyte ***ghosts*** . Escherichia coli Type I was suspended in aq. NaCl and blended. The product was centrifuged to remove cell debris. The . . by Sephadex gel column. Papain inactivation of pili was correlated with degrdn. of the 28-kilodalton protein on the pili. This ***lectin*** was coupled to CNBr Sepharose for use as mannose-specific affinity resin.

ST ***lectin*** purifn pili ***bacteria*** ; Escherichia pili
lectin purifn

IT Pili
(***lectin*** purifn. from, of ***bacteria*** , erythrocyte binding site of)

IT Escherichia coli
Klebsiella pneumoniae
Neisseria gonorrhoeae
Neisseria meningitidis
Pseudomonas aeruginosa
Salmonella
Streptococcus pneumoniae
(pili of, ***lectin*** contg. erythrocyte binding site purifn. from)

IT Agglutinins and Lectins
RL: BIOL (Biological study)
(purifn. of erythrocyte binding site-contg., of ***bacterial*** pili)

IT Erythrocyte
(***ghost*** , binding site for, on ***lectin*** of ***bacterial*** pili)

IT 3458-28-4, Mannose
RL: BIOL (Biological study)
(binding to, ***lectin*** of Escherichia coli pili specific for)

IT 9001-73-4, Papain
RL: BIOL (Biological study)
(erythrocyte binding site of ***bacterial*** pili ***lectin*** inactivation with)

IT 137-16-6 151-21-3, SDS, biological studies
RL: BIOL (Biological study)
(in ***lectin*** purifn. from ***bacterial*** pili, erythrocyte binding site in relation to)

IT 9012-36-6DP, Sepharose, cyanobromide deriv., reaction products with ***lectin*** of Escherichia pili
RL: PREP (Preparation)
(prepn. of, as mannose-specific affinity probe)

L7 ANSWER 43 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN DUPLICATE 10

AN 1987:463133 BIOSIS <>LOGINID::20091202>>

DN PREV198784108573; BA84:108573

TI EFFECT OF KUPFFER CELL PHAGOCYTOSIS OF ERYTHROCYTES AND ERYTHROCYTE

GHOSTS ON SUSCEPTIBILITY TO ENDOTOXEMIA AND ***BACTEREMIA*** .
AU LOEGERING D J [Reprint author]; COMMINS L M; MINNEAR F L; GARY L A; HILL L
A
CS DEP PHYSIOLOGY, NEIL HELLMAN MED RES BUILD, ALBANY MED COLL UNION UNIV,
ALBANY, NY 12208, USA
SO Infection and Immunity, (1987) Vol. 55, No. 9, pp. 2074-2080.
CODEN: INFIBR. ISSN: 0019-9567.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 7 Nov 1987
Last Updated on STN: 7 Nov 1987
AB The phagocytosis of erythrocytes by macrophages has previously been shown
to depress macrophage function. In this study we compared the effect of
the phagocytosis of erythrocytes and erythrocyte ***ghosts*** by
Kupffer cells on the duration of the depression of complement
receptor clearance function and host defense against endotoxemia
and ***bacteremia*** . Phagocytosis of erythrocytes and erythrocyte
ghosts was induced in rats by the injection of rat erythrocytes
or erythrocyte ***ghosts*** coated with anti-rat erythrocyte
immunoglobulin G (EIgG and GIgG, respectively). The hepatic uptake of
EIgG and GIgG (17.4 .times. 108/100 g) occurred during the first 30 min
after injection. The digestion of phagocytized EIgG and GIgG, as assessed
by electron microscopy, was complete at 24 and 3 h after injection,
respectively. The depression of Kupffer cell complement ***receptor***
clearance function caused by EIgG and GIgG returned to normal by 6 h after
injection of EIgG and by 3 h after injection of GIgG. Phagocytosis of
EIgG depressed the survival rate after endotoxemia and ***bacteremia***
when endotoxin or ***bacteria*** were injected at 30 min after EIgG.
The survival rate returned to normal when the endotoxin and
bacteria were injected at 12 and 6 h after the EIgG,
respectively.
Phagocytosis of GIgG did not depress the survival rate after endotoxemia
and ***bacteremia*** . Thus, compared with erythrocytes, erythrocyte
ghosts are more rapidly digested after phagocytosis, depress
complement ***receptor*** function for a shorter period of time, and
cause less depression of host defense. These findings indicate that the
contents of erythrocytes play an important role in the impairment of host
defense caused by the phagocytosis of erythrocytes by Kupffer cells.
TI EFFECT OF KUPFFER CELL PHAGOCYTOSIS OF ERYTHROCYTES AND ERYTHROCYTE
GHOSTS ON SUSCEPTIBILITY TO ENDOTOXEMIA AND ***BACTEREMIA*** .
AB . . . been shown to depress macrophage function. In this study we
compared the effect of the phagocytosis of erythrocytes and erythrocyte
ghosts by Kupffer cells on the duration of the depression of
complement ***receptor*** clearance function and host defense against
endotoxemia and ***bacteremia*** . Phagocytosis of erythrocytes and
erythrocyte ***ghosts*** was induced in rats by the injection of rat
erythrocytes or erythrocyte ***ghosts*** coated with anti-rat
erythrocyte immunoglobulin G (EIgG and GIgG, respectively). The hepatic
uptake of EIgG and GIgG (17.4 .times. 108/100 . . . assessed by electron
microscopy, was complete at 24 and 3 h after injection, respectively. The
depression of Kupffer cell complement ***receptor*** clearance
function caused by EIgG and GIgG returned to normal by 6 h after injection
of EIgG and by 3 h after injection of GIgG. Phagocytosis of EIgG
depressed the survival rate after endotoxemia and ***bacteremia***
when endotoxin or ***bacteria*** were injected at 30 min after EIgG.

The survival rate returned to normal when the endotoxin and ***bacteria*** were injected at 12 and 6 h after the EIgG, respectively.

Phagocytosis of GIgG did not depress the survival rate after endotoxemia and ***bacteremia***. Thus, compared with erythrocytes, erythrocyte ***ghosts*** are more rapidly digested after phagocytosis, depress complement ***receptor*** function for a shorter period of time, and cause less depression of host defense. These findings indicate that the contents. . .

IT Miscellaneous Descriptors

RAT COMPLEMENT ***RECEPTOR*** CLEARANCE DEPRESSION HOST DEFENSE IMPAIRMENT

L7 ANSWER 44 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN DUPLICATE 11

AN 1987:66161 BIOSIS <>LOGINID::20091202>>

DN PREV198783034487; BA83:34487

TI ***LECTIN*** BINDING SITES IN PARAMECIUM-TETRAURELIA CELLS II. LABELING ANALYSIS PREDOMINANTLY OF NON-SECRETORY COMPONENTS.

AU LUETHE N [Reprint author]; PLATTNER H

CS FAC BIOL, UNIV KONSTANZ, POB 5560, D-7750 KONSTANZ, W GER

SO Histochemistry, (1986) Vol. 85, No. 5, pp. 377-388.
CODEN: HCMYAL. ISSN: 0301-5564.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 24 Jan 1987

Last Updated on STN: 24 Jan 1987

AB All the ***lectin*** -FITC conjugates tested (ConA, RCA II, WGA) bind to the surface of Paramecium cells. Yet only WGA yields a distinct fluorescent pattern; it contours the basis of cilia and in some cells it brilliantly stains a few neighbouring rows of the regular surface fields in the anteroventral region (a region known to contain extensive fields of linear aggregates of freeze-fracture particles and to be engaged in conjugation). Incubation *in vivo* with WGA-FITC resulted in the selective labeling of the cytopharyngeal region as well as of the cytoproct. On Lowicryl K4M sections, WGA-gold probes concomitantly labeled disk-shaped vesicles that are assumed in the literature to serve as shuttle vesicles between these two cell regions and, thus, to connect forming and defecating digesting vacuoles (stages DV I and DV IV). On K4M sections WGA-Au stains also most other components of the lysosomal system. Also on K4M sections RCA II-Au labeled the walls of ***bacteria*** contained in DV I and II type digesting vacuoles (but not lysosomes identified bona fide by their size and shape and by their frequent vicinity to or continuity with digesting vacuoles). The WGA data largely support previous conclusions on the possible functional connection of all these elements (DV I-IV, smaller lysosomes, disk-shaped vesicles etc.) of the lysosomal system in Paramecium, as proposed by Allen and his group on the basis of other lines of evidence. As shown in the accompanying paper, ConA-FITC stained ***ghosts*** (formed after massive trichocyst exocytosis) also abort into DV-like structures. The different results obtained with the three lectins tested reflect the complex sorting machinery contained in the elaborate lysosomal system of a Paramecium cell. In the cytosol, finally, there occurs a particularly intense staining with ConA-gold, applied to Lowicryl sections, that probably represents glycogen-like particles. The same procedure reveals some weak staining of secretory contents and of nuclear structures.

TI ***LECTIN*** BINDING SITES IN PARAMECIUM-TETRAURELIA CELLS II.
LABELING ANALYSIS PREDOMINANTLY OF NON-SECRETORY COMPONENTS.

AB All the ***lectin*** -FITC conjugates tested (ConA, RCA II, WGA) bind
to the surface of Paramecium cells. Yet only WGA yields a distinct
fluorescent. . . WGA-Au stains also most other components of the
lysosomal system. Also on K4M sections RCA II-Au labeled the walls of
bacteria contained in DV I and II type digesting vacuoles (but
not

lysosomes identified bona fide by their size and shape. . . Allen and
his group on the basis of other lines of evidence. As shown in the
accompanying paper, ConA-FITC stained ***ghosts*** (formed after
massive trichocyst exocytosis) also about into DV-like structures. The
different results obtained with the three lectins tested reflect. . .

L7 ANSWER 45 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
AN 1987:191430 CAPLUS <>LOGINID::20091202>>
DN 106:191430
OREF 106:30961a,30964a
TI Characterization and ***receptor*** binding specificities of the
X-binding UTI Escherichia coli adhesin AFA-I
AU Schmidt, M. Alexander; Walz, Waltraud; Schoolnik, Gary K.
CS Sch. Med., Stanford Univ., Stanford, CA, 94305, USA
SO FEMS Symposium (1986), 31(Protein-Carbohydr. Interact. Biol. Syst.),
253-62
CODEN: FEMSDW; ISSN: 0163-9188
DT Journal
LA English
AB AFA-I, a mannose-resistant, P-independent, X-binding afimbral E. coli
adhesion was purified from a recombinant strain and chem., functionally,
and serol. characterized. AFA-I exists on the ***bacterial*** surface
and free as a macromol. aggregate in the supernatant of spent culture
medium. It is composed of a single (repeating) 16,000-dalton (D)
polypeptide subunit. The AFA-I protein amino acid compn. is remarkable
for the presence of 2.5-3.0 cysteines/subunit and for a marked decrease in
hydrophobic amino acids as compared to subunits of E. coli pili. Since
AFA-I travels as a monomer in SDS-PAGE under nonreducing conditions, no
disulfide bonds exist between subunits and .gtoreq.1 free SH/subunit is
available. The AFA-I N-terminal amino acid sequence through residue 24
was unrelated to any known E. coli fimbrial sequence. Immuno-gold
labeling demonstrated the afimbral nature of the AFA-I protein on the
bacterial cell surface. Anti-AFA-I sera bound AFA-I in Western
blots of 4 of 16 X-binding E. coli urine isolates. They did not bind MS
or P pili. HB 101 (pIL 14), the AFA-I recombinant strain, agglutinated
only human or gorilla erythrocytes, indicating a preference for
receptor mols. on the red cells of man and the anthropoid apes.
AFA-I did not bind glycoporphin A or sialyl glycosides and is therefore
distinct from the E. coli X-binding adhesins with M and S specificity.
The AFA-I ***receptor*** was found to be abundant and diffusely
distributed on HeLa tissue culture monolayer cells surface by indirect
fluorescent microscopy. Total lipid exts. of human erythrocytes and
voided uroepithelial cells proved neg. or specific binding of AFA-I. The
AFA-I protein was shown to bind to a doublet of probably peripheral
(glyco)proteins from human erythrocyte ***ghosts*** of
.apprx.96,000-98,000 mol. wt.
TI Characterization and ***receptor*** binding specificities of the
X-binding UTI Escherichia coli adhesin AFA-I
AB . . . afimbral E. coli adhesion was purified from a recombinant strain

and chem., functionally, and serol. characterized. AFA-I exists on the ***bacterial*** surface and free as a macromol. aggregate in the supernatant of spent culture medium. It is composed of a single. . . unrelated to any known E. coli fimbrial sequence. Immuno-gold labeling demonstrated the afimrial nature of the AFA-I protein on the ***bacterial*** cell surface. Anti-AFA-I sera bound AFA-I in Western blots of 4 of 16 X-binding E. coli urine isolates. They did. . . P pili. HB 101 (pIL 14), the AFA-I recombinant strain, agglutinated only human or gorilla erythrocytes, indicating a preference for ***receptor*** mols. on the red cells of man and the anthropoid apes. AFA-I did not bind glycoporphin A or sialyl glycosides and is therefore distinct from the E. coli X-binding adhesins with M and S specificity. The AFA-I ***receptor*** was found to be abundant and diffusely distributed on HeLa tissue culture monolayer cells surface by indirect fluorescent microscopy. Total. . . binding of AFA-I. The AFA-I protein was shown to bind to a doublet of probably peripheral (glyco)proteins from human erythrocyte ***ghosts*** of .apprx.96,000-98,000 mol. wt.

ST adhesion AFAI Escherichia ***receptor*** binding
 IT Escherichia coli
 (adhesin AFA-I, characterization and ***receptor*** binding of)
 IT Agglutinins and Lectins
 RL: BIOL (Biological study)
 (adhesive factors, AFA-I, of Escherichia coli, characterization and ***receptor*** binding of)

L7 ANSWER 46 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
 DUPLICATE 12

AN 1986:280942 BIOSIS <>LOGINID::20091202>>

DN PREV198682024805; BA82:24805

TI THE BINDING OF PSEUDOMONAS-AERUGINOSA OUTER MEMBRANE ***GHOSTS*** TO HUMAN BUCCAL EPITHELIAL CELLS.

AU DOIG P [Reprint author]; FRANKLIN A L; IRVIN R T

CS DEP BOTANY MICROBIOL, ERINDALE COLL, UNIV TORONTO, MISSISSAUGA, ONT, CAN L5L 1C6

SO Canadian Journal of Microbiology, (1986) Vol. 32, No. 2, pp. 160-166.
 CODEN: CJMIAZ. ISSN: 0008-4166.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 4 Jul 1986

Last Updated on STN: 4 Jul 1986

AB The binding of outer membrane (OM) ***ghosts*** derived from Pseudomonas aeruginosa strain 492c to human buccal epithelial cells (BECs) was examined. Electron microscopic examination of the binding of OM ***ghosts*** to BECs revealed direct OM ***ghost*** -BEC interaction.

Equilibrium analysis of the binding of OM ***ghosts*** to trypsinized BECs employing the Langmuir adsorption isotherm indicated the number of binding sites (N) to be 1.3 .times. 10⁻⁴ .mu.g protein per BEC with an apparent association constant (Ka) of 3.4 .times. 10⁻² mL/.mu.g protein. The Langmuir analysis of binding of OM ***ghosts*** to untrypsinized BECs was complex, suggesting two possible classes of receptors, a high affinity-low copy number class (Ka, 7.8 .times. 10⁻² mL/.mu.g protein; N, 8.6 .times. 10⁻⁵ .mu.g protein per BEC) and a low affinity-high copy number class (Ka, 3.7 .times. 10⁻³ mL/.mu.g protein; N, 9.2 .times. 10⁻⁴ .mu.g protein per BEC). Sugar inhibition studies incorporating

D-galactose enhanced binding to each BEC type. N-Acetylneurameric acid and N-acetylgalcosamine both enhanced binding of OM ***ghosts*** to trypsinized BECs, while inhibiting binding to trypsinized BECs. D-Arabinose inhibited binding to both BEC types. Binding of OM ***ghosts*** to both BEC types was greatly inhibited by D-fucose, while L-fucose only greatly inhibited binding to trypsinized BECs. These sugar inhibition data demonstrated a difference in the binding of OM ***ghosts*** to trypsinized and untrypsinized BECs and possibly reveal the nature of ***receptor*** (s), free of possible ***bacterial*** metabolic effects. These data indicated that OM ***ghosts*** from 492c appear to bind to BECs in a similar manner to the intact ***bacteria*** and represent a simple model system to study the adhesion

of *P. aeruginosa* to BECs.

TI THE BINDING OF PSEUDOMONAS-AERUGINOSA OUTER MEMBRANE ***GHOSTS*** TO HUMAN BUCCAL EPITHELIAL CELLS.

AB The binding of outer membrane (OM) ***ghosts*** derived from *Pseudomonas aeruginosa* strain 492c to human buccal epithelial cells (BECs) was examined. Electron microscopic examination of the binding of OM ***ghosts*** to BECs revealed direct OM ***ghost*** -BEC interaction.

Equilibrium analysis of the binding of OM ***ghosts*** to trypsinized BECs employing the Langmuir adsorption isotherm indicated the number of binding sites (N) to be 1.3 .times. 10⁻⁴ . . . BEC with an apparent association constant (Ka) of 3.4 .times. 10⁻² mL/.μg protein. The Langmuir analysis of binding of OM ***ghosts*** to untrypsinized BECs was complex, suggesting two possible classes of receptors, a high affinity-low copy number class (Ka, 7.8 .times.. . . Sugar inhibition studies incorporating D-galactose enhanced binding to each BEC type. N-Acetylneurameric acid and N-acetylgalcosamine both enhanced binding of OM ***ghosts*** to untrypsinized BECs, while inhibiting binding to trypsinized BECs. D-Arabinose inhibited binding to both BEC types. Binding of OM ***ghosts*** to both BEC types was greatly inhibited by D-fucose, while L-fucose only greatly inhibited binding to untrypsinized BECs. These sugar inhibition data demonstrated a difference in the binding of OM ***ghosts*** to trypsinized and untrypsinized BECs and possibly reveal the nature of ***receptor*** (s), free of possible ***bacterial*** metabolic effects. These data indicated that OM ***ghosts*** from 492c appear to bind to BECs in a similar manner to

the intact ***bacteria*** and represent a simple model system to study the adhesion of *P. aeruginosa* to BECs.

ORGN Classifier

Pseudomonadaceae 06508

Super Taxa

Gram-Negative Aerobic Rods and Coccii; Eubacteria; ***Bacteria*** ;

Microorganisms

Taxa Notes

Bacteria , Eubacteria, Microorganisms

L7 ANSWER 47 OF 68 LIFESCI COPYRIGHT 2009 CSA on STN

AN 86:72100 LIFESCI <>LOGINID:20091202>>

TI Studies on the staphylococcal alpha-toxin ***receptor*** on myelin and RB-RBC.

BACTERIAL PROTEIN TOXINS.

AU Harshman, S.; Sugg, N.; Falmagne, P. [editor]; Alouf, J.E. [editor]; Fehrenbach, F.J. [editor]; Jeljaszewicz, J. [editor]; Thelestam, M.

[editor]
 CS Dep. Microbiol., Vanderbilt Univ. Sch. Med., Nashville, TN 37232, USA
 SO ZENTRALBL. BAKTERIOL. MIKROBIOL. HYG., (1986) pp. 213-220.
 Meeting Info.: 2. European Workshop on Bacterial Protein Toxins. Wepion (Belgium). 30 Jun-4 Jul 1985.
 ISBN: 3-437-11083-7.
 DT Book
 TC Conference
 FS J; M
 LA English
 AB Staphylococcal alpha-toxin is an extracellular protein that is produced by most pathogenic strains of *Staphylococcus aureus*. It is selectively hemolytic, induces spastic paralysis in smooth muscle, provokes dermal necrosis, and is lethal for most laboratory animals. Although the detailed molecular mechanism of its lethal activity is not known, several lines of evidence led to the conclusion that the central or peripheral nervous tissue is the critical target organ. Both Schwann cell generated myelin of peripheral tissue and oligodendroglial cell generated myelin of central nervous tissue are susceptible to selective disruption by alpha-toxin. The authors report here recent data that suggests that a common lipoprotein exists in the membranes of myelin and Rb-rbc ***ghosts*** that may function as the specific alpha-toxin ***receptor***.
 TI Studies on the staphylococcal alpha-toxin ***receptor*** on myelin and RB-RBC.
 BACTERIAL PROTEIN TOXINS.
 AB . . . The authors report here recent data that suggests that a common lipoprotein exists in the membranes of myelin and Rb-rbc ***ghosts*** that may function as the specific alpha-toxin ***receptor***.

L7 ANSWER 48 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 1984:486921 CAPLUS <<LOGINID::20091202>>
 DN 101:86921
 OREF 101:13301a,13304a
 TI Specific binding assays utilizing analyte-cytolysin conjugates
 IN Freytag, William J.; Litchfield, William John
 PA du Pont de Nemours, E. I., and Co. , USA
 SO Eur. Pat. Appl., 48 pp.
 CODEN: EPXXDW
 DT Patent
 LA English
 FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------|---|------|----------|-----------------|----------|
| PI | EP 106370 | A2 | 19840425 | EP 1983-110469 | 19831020 |
| | EP 106370 | A3 | 19860226 | | |
| | R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE | | | | |
| | US 4517303 | A | 19850514 | US 1982-435455 | 19821020 |
| | CA 1206899 | A1 | 19860701 | CA 1983-439211 | 19831018 |
| | DK 8304811 | A | 19840421 | DK 1983-4811 | 19831019 |
| | JP 59094069 | A | 19840530 | JP 1983-194473 | 19831019 |
| PRAI | US 1982-435455 | A | 19821020 | | |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB A rapid and sensitive membrane lytic assay is described for the detn. of low concns. (micromolar to picomolar range) of analytes (e.g., drugs, metabolites, hormones, pesticides, food toxins, viruses, cancer cell markers) in a liq. medium by using a new analyte deriv.-cytolysin conjugate, an analyte-specific binding agent (e.g., antibody, hormone

receptor , ***lectin*** , specific binding protein), and vesicles (e.g., lipid vesicles, erythrocytes, or their ***ghosts***) contg. detectable markers (enzymes, cofactors, chromophores, fluorophores, ions, spin labels). Uncombined conjugate alters the permeability of the vesicles, resulting in release and quantitation of the marker which is correlated to the concn. of analyte initially present. Thus, digoxin was detd. by a homogeneous immunoassay by using antidigoxin antibodies purified by affinity chromatog., ouabain-melittin conjugate, and lipid vesicles with sequestered alk. phosphatase. The absorbance of the soln. was monitored continuously at 410 nm.

OSC.G 22 THERE ARE 22 CAPLUS RECORDS THAT CITE THIS RECORD (22 CITINGS)

AB . . . cell markers) in a liq. medium by using a new analyte deriv.-cytolysin conjugate, an analyte-specific binding agent (e.g., antibody, hormone ***receptor*** , ***lectin*** , specific binding protein), and vesicles (e.g., lipid vesicles, erythrocytes, or their ***ghosts***) contg. detectable markers (enzymes, cofactors, chromophores, fluorophores, ions, spin labels). Uncombined conjugate alters the permeability of the vesicles, resulting in. . .

IT ***Bacteria***
(surface markers of, detn. of, by membrane lysis assay)

L7 ANSWER 49 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN DUPLICATE 13

AN 1984:317890 BIOSIS <>LOGINID::20091202>>

DN PREV198478054370; BA78:54370

TI PHYSIOLOGICAL RESPONSES OF BACILLUS SPECIES TO CONCANAVALIN A 1. BINDING OF CONCANAVALIN A TO BACILLUS-CEREUS ATCC-14579 AND BACILLUS-LICHENIFORMIS IFO-12107.

AU CHAN K-Y [Reprint author]; LAU T-M
CS DEP BIOL, CHIN UNIV HONG KONG, SHATIN, NT, HONG KONG
SO Microbios, (1984) Vol. 39, No. 156, pp. 121-128.
CODEN: MCBIA7. ISSN: 0026-2633.

DT Article

FS BA

LA ENGLISH

AB The binding of labeled concanavalin A (Con A), a mitogenic protein, to cells of *B. cereus* ATCC 14579 and *B. licheniformis* IFO 12107 indicated that almost identical levels of 3H-Con A were located in the whole cell, and the protoplast and the membrane ***ghost*** fractions, evidence that Con A did not bind to the teichoic acids and peptidoglycans of the cell walls, but only to the membrane teichoic acids. The binding of 3H-Con A to the cells was temperature-dependent. Greater amounts of H-Con A bound with various cell fractions at 35.degree. C than at 0.degree. C. Evidently, at 35.degree. C Con A not only bound to the specific ***receptor*** sites of the cells but also bound to the cell envelope

by non-specific binding which occurred mainly on the surface of the ***bacterial*** cells. That only negligible levels of 3H-Con A bound with the cytoplasm fractions suggested that the cells were unable to transport Con A molecules into the cytoplasm.

AB. . . indicated that almost identical levels of 3H-Con A were located in the whole cell, and the protoplast and the membrane ***ghost*** fractions, evidence that Con A did not bind to the teichoic acids and peptidoglycans of the cell walls, but only. . . fractions at 35.degree. C than at 0.degree. C. Evidently, at 35.degree. C Con A not only bound to the specific ***receptor*** sites of the cells but also bound to the cell envelope by non-specific binding which occurred mainly on the

surface of the ***bacterial*** cells. That only negligible levels of 3H-Con A bound with the cytoplasm fractions suggested that the cells were unable to. . .

ORGN Classifier

Endospore-forming Gram-Positives 07810

Super Taxa

Eubacteria; ***Bacteria*** ; Microorganisms

Taxa Notes

Bacteria , Eubacteria, Microorganisms

L7 ANSWER 50 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN

AN 1983:277110 BIOSIS <>LOGINID::20091202>

DN PREV198376034602; BA76:34602

TI AGGREGATION OF HUMAN PLATELETS AND ADHESION OF STREPTOCOCCUS-SANGUIS.

AU HERZBERG M C [Reprint author]; BRINTZENHOE K L; CLAWSON C C

CS SCH DENT, UNIV MINN, MINNEAPOLIS, MINN 55455, USA

SO Infection and Immunity, (1983) Vol. 39, No. 3, pp. 1457-1469.
CODEN: INFIBR. ISSN: 0019-9567.

DT Article

FS BA

LA ENGLISH

AB The hypothesis that human platelets selectively bind or adhere to strains of *S. Sanguis* and *S. mutans* and aggregate, as a result, into an *in vitro* thrombus was investigated. Adhesion was uncoupled from activation and aggregation by incubating streptococci with platelet ***ghosts*** in a simple, quantitative assay. Adhesion was mediated by protease-sensitive components on the streptococci and platelet ***ghosts*** rather than cell surface carbohydrates or dextrans, plasma components or divalent cations. The same streptococci were also studied by standard aggregometry techniques. Platelet-rich plasma was activated and aggregated by certain isolates of *S. sanguis*. Platelet ***ghosts*** bound the same strains selectively under Ca²⁺ and plasma-depleted conditions. Fresh platelets activated after washing; Ca²⁺ had to be restored. Aggregation required fresh platelets in Ca²⁺-restored plasma and was inducible by washed streptococcal cell walls. These reactions in the binding and aggregometry assays were confirmed by transmission EM. Surface microfibrils on intact *S. sanguis* were identified. These appendages bound *S. sanguis* to platelets. The selectivity of adhesion of the various *S. sanguis* strains to platelet ***ghosts*** or Ca²⁺- and plasma-depleted fresh washed platelets was similar for all donors. Thus, the platelet binding site was expressed widely in the population and was unlikely to be an artifact of membrane aging or preparation. Since selective adhesion of *S. sanguis* to platelets was apparently required for aggregation, functionally defined receptors for ligands on certain strains of *S. sanguis* may be present on human platelets. Some differences in the selectivity and rate of the aggregation response were noted among platelet donors; the meaning of the variability requires further study. These interactions may contribute to platelet accretion in the initiation and development of vegetative lesions in subacute ***bacterial*** endocarditis.

AB. . . result, into an *in vitro* thrombus was investigated. Adhesion was uncoupled from activation and aggregation by incubating streptococci with platelet ***ghosts*** in a simple, quantitative assay. Adhesion was mediated by protease-sensitive components on the streptococci and platelet ***ghosts*** rather than cell surface carbohydrates or dextrans, plasma components or divalent cations. The same streptococci were also studied by standard aggregometry techniques. Platelet-rich plasma was activated

and aggregated by certain isolates of *S. sanguis*. Platelet ***ghosts*** bound the same strains selectively under Ca²⁺ and plasma-depleted conditions. Fresh platelets activated after washing; Ca²⁺ had to be restored.. . . identified. These appendages bound *S. sanguis* to platelets. The selectivity of adhesion of the various *S. sanguis* strains to platelet ***ghosts*** or Ca²⁺- and plasma-depleted fresh washed platelets was similar for all donors. Thus, the platelet binding site was expressed widely. . . requires further study. These interactions may contribute to platelet accretion in the initiation and development of vegetative lesions in subacute ***bacterial*** endocarditis.

IT Miscellaneous Descriptors

STREPTOCOCCUS-MUTANS HUMAN PLATELET ***GHOSTS*** PROTEASE SENSITIVE COMPONENTS CALCIUM ION DEPLETION PLASMA DEPLETION SURFACE MICRO FIBRILS ***LIGAND*** RECEPTORS ACCRETION VEGETATIVE LESIONS SUBACUTE ***BACTERIAL*** ENDO CARDITIS

ORGN Classifier

Gram-Positive Coccii 07700

Super Taxa

Eubacteria; ***Bacteria*** ; Microorganisms

Taxa Notes

Bacteria , Eubacteria, Microorganisms

ORGN Classifier

Hominidae 86215

Super Taxa

Primates; Mammalia; Vertebrata; Chordata; Animalia

Taxa Notes

Animals, Chordates, Humans, Mammals, Primates,.. . .

L7 ANSWER 51 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

AN 1983:245983 BIOSIS <>LOGINID::20091202>

DN PREV198376003475; BA76:3475

TI MICRO INJECTION OF MACRO MOLECULES INTO NORMAL MURINE LYMPHOCYTES BY MEANS OF CELL FUSION 2. ENHANCEMENT AND SUPPRESSION OF MITOGENIC RESPONSES BY MICRO INJECTION OF MONO CLONAL ANTI CYCLIC AMP INTO B LYMPHOCYTES.

AU OHARA J [Reprint author]; SUGI M; FUJIMOTO M; WATANABE T

CS DEP OF IMMUNOL, SAGA MED SCH, NABESHIMA, SAGA 840-01, JPN

SO Journal of Immunology, (1982) Vol. 129, No. 3, pp. 1227-1232.

CODEN: JOIMA3. ISSN: 0022-1767.

DT Article

FS BA

LA ENGLISH

AB Reproducible methods are now available for introducing protein molecules such as antibodies into normal murine lymphocytes by fusion with protein molecule-containing erythrocyte ***ghosts***. Monoclonal antibodies against cAMP were raised by hybridoma technique and packed into erythrocyte ***ghosts***. Then, monoclonal anti-cAMP containing ***ghosts*** were fused with splenic B lymphocytes by polyethylene glycol-mediated fusion at various intervals after LPS stimulation. This method made it possible to quantitatively microinject antibodies into B lymphocytes. Microinjection of anti-cAMP antibody molecules into lymphocytes at a very early stage of LPS [lipopolysaccharide] stimulation resulted in a marked enhancement of DNA synthetic responses and increased numbers of plaque-forming cells. Intracellular cAMP levels were markedly decreased after microinjection of monoclonal anti-cAMP, suggesting that lowering the intracellular cAMP level in the B lymphocytes at an early stage of stimulation might have induced the enhanced proliferative and

differentiative responses to LPS. Similar enhancing effects on cell proliferation were obtained when antibodies were injected 18 h after stimulation. Microinjection of anti-cAMP at 12 h after culture inhibited the DNA synthetic responses, and induction of plaque-forming cells was suppressed when anti-cAMP was injected 6 h after LPS stimulation. The data suggest the biphasic regulatory roles of cAMP at the early stage of B lymphocyte activation. This approach may be useful in identifying regulatory molecules in B lymphocytes induced by mitogenic or antigenic stimulation.

AB. . . are now available for introducing protein molecules such as antibodies into normal murine lymphocytes by fusion with protein molecule-containing erythrocyte ***ghosts***. Monoclonal antibodies against cAMP were raised by hybridoma technique and packed into erythrocyte ***ghosts***. Then, monoclonal anti-cAMP containing ***ghosts*** were fused with splenic B lymphocytes by polyethylene glycol-mediated fusion at various intervals after LPS stimulation. This method made it. . .

IT Miscellaneous Descriptors

PLAQUE FORMING CELL LIPO POLY ***SACCHARIDE*** DNA SYNTHESIS

ORGN Classifier

Bacteria 05000

Super Taxa

Microorganisms

Taxa Notes

Bacteria, Eubacteria, Microorganisms

ORGN Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates,. . .

L7 ANSWER 52 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

AN 1982:237843 BIOSIS <>LOGINID::20091202>

DN PREV198274010323; BA74:10323

TI MICRO INJECTION OF MACRO MOLECULES INTO NORMAL MURINE LYMPHOCYTES BY CELL FUSION TECHNIQUE 1. QUANTITATIVE MICRO INJECTION OF ANTIBODIES INTO NORMAL SPLENIC LYMPHOCYTES.

AU OHARA J [Reprint author]; WATANABE T

CS DEP IMMUNOL, SAGA MED SCH, NABESHIMA, SAGA 840-01, JAPAN

SO Journal of Immunology, (1982) Vol. 128, No. 3, pp. 1090-1096.

CODEN: JOIMAA3. ISSN: 0022-1767.

DT Article

FS BA

LA ENGLISH

AB Human erythrocyte ***ghosts*** loaded with various kinds of protein molecules were fused with mouse splenic lymphocytes by means of polyethylene glycol supplemented with poly-L-arginine and dimethylsulfoxide. This fusion method made quantitative microinjection of IgG and other proteins into intact lymphocytes possible. The injection itself did not alter cell viability and lymphocytes given protein molecules retained intact response activity when they were stimulated with mitogens. Rabbit anti-cAMP was purified by affinity chromatography and injected into lymphocytes. Antibody activity in the cell lysates was measured by using ¹²⁵I-labeled cAMP as an antigen and it was shown that antibody molecules were quantitatively injected and immunologically active

in the cells. Antigen binding activity of anti-cAMP antibodies in the nonstimulated lymphocytes was stable and intact even 24 h after microinjection, whereas the activity rapidly decreased in mitogen-stimulated lymphocytes, indicating that some immunologic or enzymatic mechanisms for inactivating antibodies were induced in mitogen-stimulated cells. Microinjection of anti-cAMP markedly enhanced the proliferative responses of lymphocytes to mitogens such as concanavalin A or lipopolysaccharide and reversed the effect of a known elevator of intracellular cAMP. These observations have implications for the role of cAMP in early lymphocyte activation events.

AB Human erythrocyte ***ghosts*** loaded with various kinds of protein molecules were fused with mouse splenic lymphocytes by means of polyethylene glycol supplemented with . . .

IT Miscellaneous Descriptors

HUMAN ERYTHROCYTE ***GHOST*** RABBIT ANTIGEN BINDING ACTIVITY
LYMPHOCYTE ACTIVATION CYCLIC AMP CONCANAVALIN A LIPO POLY
SACCHARIDE

ORGN Classifier

Bacteria 05000

Super Taxa

Microorganisms

Taxa Notes

Bacteria , Eubacteria, Microorganisms

ORGN Classifier

Leguminosae 26260

Super Taxa

Dicotyledones; Angiospermae; Spermatophyta; Plantae

Taxa Notes

Angiosperms, Dicots, Plants, Spermatophytes, Vascular Plants

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STN DUPLICATE 14

AN 1982:198350 BIOSIS <>LOGINID::20091202>>

DN PREV198273058334; BA73:58334

TI FAST RESPONSES OF ***BACTERIAL*** MEMBRANES TO VIRUS ADSORPTION A FLUORESCENCE STUDY.

AU BAYER M E [Reprint author]; BAYER M H

CS INST FOR CANCER RES, FOX CHASE CANCER CENT, PHILADELPHIA, PA 19111, USA

SO Proceedings of the National Academy of Sciences of the United States of America, (1981) Vol. 78, No. 9, pp. 5618-5622.

CODEN: PNASA6. ISSN: 0027-8424.

DT Article

FS

LA ENGLISH

AB After collision with their host cells, virus particles may remain mobile on cell surfaces until they become attached at firm binding sites. It is proposed that a virion will arrive within a typical median time at such a site, generating a membrane signal such as an increased membrane fluorescence in cells labeled with the voltage-sensitive dyes 8-anilino-1-naphthalene-sulfonate (Mg-salt) (ANS), N-phenylnaphthylamine (NPA) or 3,3'-dipentyl-2,2'-oxacarboxyanine (di-O-C5[3]). The time span between virus adsorption and fluorescence response varies widely among phages and also depends on ***bacterial*** [Escherichia coli, *Salmonella* ado] strain, metabolic state and type of dye. di-O-C5[3]-labeled cells react within 1 s to uncouplers such as carbonyl cyanide m-chlorophenylhydrazone (CCCP). Cells labeled with ANS and NPA react to CCCP in 4-6 s. ***Bacteriophages*** (T4, T5, .chi., and

BF23), added to ANS-labeled cells, change the fluorescence in 9-15 s. T-even ***ghosts*** cause a response at identical times. Baseplate-defective phage mutant T412- and isolated adsorption organelles of smaller viruses fail to cause an effect. di-O-C5[3]-labeled cells respond to T4 at a multiplicity of infection .gt;req. 40 within 1 s. A longer time (8 s) is required at lower multiplicities. The ***receptor*** -degrading phages .epsilon.15, .epsilon.34, c341 and K29 need the longest time (1 min for ANS) to cause a fluorescence increase. The delayed fluorescence response may be concomitant with the surface walk of the virion which is terminated at an injection site. T4 tail sheath contraction coincided with the onset of the membrane fluorescence response.

- TI FAST RESPONSES OF ***BACTERIAL*** MEMBRANES TO VIRUS ADSORPTION A FLUORESCENCE STUDY.
- AB. . . or 3,3'-dipentyl-2,2'-oxacarbocyanine (di-O-C5[3]). The time span between virus adsorption and fluorescence response varies widely among phages and also depends on ***bacterial*** [Escherichia coli, Salmonella ado] strain, metabolic state and type of dye. di-O-C5[3]-labeled cells react within 1 s to uncouplers such as carbonyl cyanide m-chlorophenylhydrazone (CCCP). Cells labeled with ANS and NPA react to CCCP in 4-6 s. ***Bacteriophages*** (T4, T5, .chi., and BF23), added to ANS-labeled cells, change the fluorescence in 9-15 s. T-even ***ghosts*** cause a response at identical times. Baseplate-defective phage mutant T412- and isolated adsorption organelles of smaller viruses fail to cause. . . a multiplicity of infection .gt;req. 40 within 1 s. A longer time (8 s) is required at lower multiplicities. The ***receptor*** -degrading phages .epsilon.15, .epsilon.34, c341 and K29 need the longest time (1 min for ANS) to cause a fluorescence increase. The. . .
- IT Miscellaneous Descriptors
ESCHERICHIA-COLI SALMONELLA-ADO PHAGE T-4 PHAGE T-5 T EVEN PHAGE
GHOSTS PHAGE CHI PHAGE BF-23 ***RECEPTOR*** DEGRADING
- PHAGE
EPSILON 15 PHAGE EPSILON 34 PHAGE C-341 PHAGE K-29 8 ANILINO-1
NAPHTHALENESULFONATE N PHENYL NAPHTHYLAMINE 3 3' DI. . .
- ORGN . . .
Viruses; Microorganisms
Taxa Notes
Double-Stranded DNA Viruses, Microorganisms, Viruses
- ORGN Classifier
Enterobacteriaceae 06702
Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria ; Microorganisms
Taxa Notes
Bacteria , Eubacteria, Microorganisms
- ORGN Classifier
Hepaticae 21400
Super Taxa
Bryophyta; Plantae
Taxa Notes
Bryophytes, Nonvascular Plants, Plants
- L7 ANSWER 54 OF 68 MEDLINE on STN
AN 1981266179 MEDLINE <>LOGINID::20091202>>
DN PubMed ID: 6790668
TI Adsorption of the defective phage PBS Z1 to Bacillus subtilis 168 Wt.

AU Steensma H Y
SO The Journal of general virology, (1981 Jan) Vol. 52, No. Pt 1, pp. 93-101.
Journal code: 0077340. ISSN: 0022-1317.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198110
ED Entered STN: 16 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 25 Oct 1981
AB Three aspects of the adsorption of the defective phage PBS Z1 to *Bacillus subtilis* 168 Wt have been investigated. These are the kinetics, the number of receptors on the cell wall and the character of these receptors. The reaction constants for the binding of phages onto receptors, for the dissociation of the phage- ***receptor*** complex and for the transition from reversible to irreversible binding of the phages were calculated from adsorption curves obtained by an enzyme-linked immunosorbent assay (ELISA). They were $1.8 \times 10(-13)$, $6.7 \times 10(-2)$ and $9.0 \times 10(-3)$ respectively. The maximum number of phages adsorbed per cell was 2700, a number limited by the surface area of the cells. Apart from the receptors on the cell wall, receptors on the cell membrane were found. This was concluded from additional adsorption experiments with stable L-forms and contracted phages. Based on these results, together with data from the literature on ***bacteriocins***, phage ***ghosts*** and yeast killer factors, a hypothesis concerning the first stage of killing by defective phages has been formulated.
AB . . . the character of these receptors. The reaction constants for the binding of phages onto receptors, for the dissociation of the phage- ***receptor*** complex and for the transition from reversible to irreversible binding of the phages were calculated from adsorption curves obtained by. . . additional adsorption experiments with stable L-forms and contracted phages. Based on these results, together with data from the literature on ***bacteriocins***, phage ***ghosts*** and yeast killer factors, a hypothesis concerning the first stage of killing by defective phages has been formulated.
CT Adsorption
*Bacillus subtilis: ME, metabolism
 Bacteriophages: ME, metabolism
Cell Membrane: AN, analysis
Cell Wall: AN, analysis
Kinetics
Receptors, Virus: AN, analysis
*Receptors, Virus: ME, metabolism
L7 ANSWER 55 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN
AN 1979:247556 BIOSIS <<LOGINID::20091202>>
DN PREV197968050060; BA68:50060
TI STRUCTURAL AND BIOCHEMICAL EXAMINATION OF ***GHOSTS*** DERIVED FROM A DEEP ROUGH HEPTOSE DEFICIENT LIPO POLY ***SACCHARIDE*** STRAIN AND A SMOOTH STRAIN OF *ESCHERICHIA-COLI*.
AU IRVIN R T [Reprint author]; LAM J; COSTERTON J W
CS DEP BIOL, UNIV CALGARY, CALGARY, ALBERTA T2N 1N4, CAN
SO Canadian Journal of Microbiology, (1979) Vol. 25, No. 4, pp. 436-446.
CODEN: CJMIAZ. ISSN: 0008-4166.
DT Article

FS BA
LA ENGLISH
AB Outer membrane derived ***ghosts*** can be readily generated from smooth and deep rough (heptose-deficient LPS [lipopolysaccharide]) strains of *E. coli* 08. Morphological and biochemical studies confirmed that ***ghosts*** of both strains are composed of protein (4 major proteins),
LPS and phospholipid (cardiolipin and phosphatidylethanolamine) in the form of a single membrane of roughly the same shape as intact normal cells. The ***ghost*** membrane cleaves only slightly in freeze-etch preparations of ***ghosts*** derived from the smooth strain as compared to the extensive cleavage plane of ***ghosts*** derived from the rough strain. The asymmetrical distribution of ***ghost*** proteins was visualized, by critical point drying and shadowing with Pt, as a relatively smooth outer surface with some discernible particles (10-15 nm) and an extremely particulate inner surface (10-15 nm particles). ***Ghosts*** derived from the smooth strain retained their structure following chloroform-methanol extraction, while ***ghosts*** derived from the rough strain fragmented with chloroform-methanol extraction. LPS-protein interactions and protein-protein interactions are apparently significant in maintaining the ***ghost*** structure.
TI STRUCTURAL AND BIOCHEMICAL EXAMINATION OF ***GHOSTS*** DERIVED FROM A DEEP ROUGH HEPTOSE DEFICIENT LIPO POLY ***SACCHARIDE*** STRAIN AND A SMOOTH STRAIN OF *ESCHERICHIA-COLI*.
AB Outer membrane derived ***ghosts*** can be readily generated from smooth and deep rough (heptose-deficient LPS [lipopolysaccharide]) strains of *E. coli* 08. Morphological and biochemical studies confirmed that ***ghosts*** of both strains are composed of protein (4 major proteins),
LPS and phospholipid (cardiolipin and phosphatidylethanolamine) in the form of a single membrane of roughly the same shape as intact normal cells. The ***ghost*** membrane cleaves only slightly in freeze-etch preparations of ***ghosts*** derived from the smooth strain as compared to the extensive cleavage plane of ***ghosts*** derived from the rough strain. The asymmetrical distribution of ***ghost*** proteins was visualized, by critical point drying and shadowing with Pt, as a relatively smooth outer surface with some discernible particles (10-15 nm) and an extremely particulate inner surface (10-15 nm particles). ***Ghosts*** derived from the smooth strain retained their structure following chloroform-methanol extraction, while ***ghosts*** derived from the rough strain fragmented with chloroform-methanol extraction. LPS-protein interactions and protein-protein interactions are apparently significant in maintaining the ***ghost*** structure.

ORGN Classifier
Enterobacteriaceae 06702
Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria ; Microorganisms
Taxa Notes
Bacteria , Eubacteria, Microorganisms

L7 ANSWER 56 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 1979:215069 BIOSIS <>LOGINID::20091202>>
DN PREV197968017573; BA68:17573

- TI BETA ADRENERGIC ***RECEPTOR*** AGONISTS INCREASE PHOSPHO LIPID METHYLATION MEMBRANE FLUIDITY AND BETA ADRENERGIC ***RECEPTOR*** ADENYLATE CYCLASE EC-4.6.1.1 COUPLING.
- AU HIRATA F [Reprint author]; STRITTMATTER W J; AXELROD J
- CS LAB CLIN SCI, NATL INST MENT HEALTH, BETHESDA, MD 20014, USA
- SO Proceedings of the National Academy of Sciences of the United States of America, (1979) Vol. 76, No. 1, pp. 368-372.
CODEN: PNASA6. ISSN: 0027-8424.
- DT Article
- FS BA
- LA ENGLISH
- AB The .beta.-adrenergic agonist L-isoproterenol stimulated the enzymic synthesis of phosphatidyl-N-monomethylethanolamine and phosphatidylcholine in rat reticulocyte ***ghosts*** containing the methyl donor S-adenosyl-L-methionine. The stimulation was stereospecific, dose-dependent, and inhibited by .beta.-adrenergic agonist propranolol. The addition of GTP inside the resealed ***ghosts*** shifted the dose-response of phospholipid methylation by L-isoproterenol to the left by 2 orders of magnitude. Direct stimulation of adenylate cyclase [ATP pyrophosphate-lyase (cyclizing), EC 4.6.1.1] with NaF or cholera toxin did not increase the methylation of phospholipids. At a concentration of S-adenosyl-L-methionine that stimulates synthesis of phosphatidyl-N-monomethylethanolamine, the activity of sioproterenol-sensitive adenylate cyclase was increased 2-fold without changes in the basal activity of adenylate cyclase and the number of .beta.-adrenergic receptors. The increase of phospholipid methylation by L-isoproterenol decreased membrane viscosity and increased translocation of methylated lipids. These findings indicate that enhancement of phospholipid methylation by L-isoproterenol decreases membrane microviscosity and thus increases lateral movement of the .beta.-adrenergic receptors and coupling with adenylate cyclase.
- TI BETA ADRENERGIC ***RECEPTOR*** AGONISTS INCREASE PHOSPHO LIPID METHYLATION MEMBRANE FLUIDITY AND BETA ADRENERGIC ***RECEPTOR*** ADENYLATE CYCLASE EC-4.6.1.1 COUPLING.
- AB The .beta.-adrenergic agonist L-isoproterenol stimulated the enzymic synthesis of phosphatidyl-N-monomethylethanolamine and phosphatidylcholine in rat reticulocyte ***ghosts*** containing the methyl donor S-adenosyl-L-methionine. The stimulation was stereospecific, dose-dependent, and inhibited by .beta.-adrenergic agonist propranolol. The addition of GTP inside the resealed ***ghosts*** shifted the dose-response of phospholipid methylation by L-isoproterenol to the left by 2 orders of magnitude. Direct stimulation of adenylate cyclase. . .
- ORGN Classifier
Vibrionaceae 06704
Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria ; Microorganisms
Taxa Notes
Bacteria , Eubacteria, Microorganisms
- ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates,. . .

STN
AN 1979:265819 BIOSIS <>LOGINID::20091202>>
DN PREV197968068323; BA68:68323
TI EFFECT OF FLUORIDE AND 5 GUANYLYL IMIDO DI PHOSPHATE ON CHOLERA TOXIN TREATED CELL.
AU GANGULY U [Reprint author]; GREENOUGH W B III
CS CHOLER RES CENT, CALCUTTA 700 016, W BENGAL, INDIA
SO Indian Journal of Experimental Biology, (1978) Vol. 16, No. 12, pp. 1271-1273.
CODEN: IJEBAA. ISSN: 0019-5189.
DT Article
FS BA
LA ENGLISH
AB Cholera toxin stimulates [rat] fat cell adenylylate cyclase after binding to a specific ganglioside (GM1) ***receptor*** . Epinephrine stimulates fat cell adenylylate cyclase and this response is enhanced by prior exposure of cells to cholera toxin. Adenylylate cyclase is stimulated by F- in a different way from hormones or cholera toxin. F- decreased the response to toxin and epinephrine separately and together at concentrations of more than 1 mM. At 25.degree. C the direct stimulating effects of F- are minimal and the blocking action is manifest. 5'-Guanylyl-imidodiphosphate (Gpp(NH)p) caused no further stimulation of basal or epinephrine-responsive adenylylate cyclase in toxin-pre-treated fat cell ***ghosts*** . Since F- blocks stimulation by epinephrine and cholera toxin, a common pathway is shared despite differing receptors. Cells treated with cholera toxin or Gpp(NH)p enhance response to epinephrine and cells pre-treated with toxin do not respond further to Gpp(NH)p, suggesting a shared regulator pathway between toxin and a guanylyl nucleotide.
AB Cholera toxin stimulates [rat] fat cell adenylylate cyclase after binding to a specific ganglioside (GM1) ***receptor*** . Epinephrine stimulates fat cell adenylylate cyclase and this response is enhanced by prior exposure of cells to cholera toxin. Adenylylate . . . blocking action is manifest. 5'-Guanylyl-imidodiphosphate (Gpp(NH)p) caused no further stimulation of basal or epinephrine-responsive adenylylate cyclase in toxin-pre-treated fat cell ***ghosts*** . Since F- blocks stimulation by epinephrine and cholera toxin, a common pathway is shared despite differing receptors. Cells treated with . . .
ORGN Classifier
 Vibrionaceae 06704
Super Taxa
 Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
 Bacteria ; Microorganisms
Taxa Notes
 Bacteria , Eubacteria, Microorganisms
ORGN Classifier
 Muridae 86375
Super Taxa
 Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
 Animals, Chordates, Mammals, Nonhuman Vertebrates,. . .
L7 ANSWER 58 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
 STN
AN 1979:149053 BIOSIS <>LOGINID::20091202>>
DN PREV197967029053; BA67:29053
TI ROLE OF CHOLESTEROL IN THE ACTION OF CEREOLYSIN ON MEMBRANES.

AU COWELL J L [Reprint author]; BERNHEIMER A W
CS DIV BACT PROD, BUR BIOL, FOOD DRUG ADM, BETHESDA, MD 20014, USA
SO Archives of Biochemistry and Biophysics, (1978) Vol. 190, No. 2, pp.
603-610.
CODEN: ABBIA4. ISSN: 0003-9861.
DT Article
FS BA
LA ENGLISH

AB The following evidence supports the concept that cholesterol in membranes is the ***receptor*** and target site for the cytolytic action of [Bacillus cereus] cereolysin. Of the various phospholipids, gangliosides and steroids tested, only cholesterol and closely related sterols (sitosterol and dihydrocholesterol) significantly inhibited the hemolytic activity of cereolysin. Acholeplasma laidlawii cells grown in the presence of cholesterol inhibited the hemolytic activity of cereolysin, but A. laidlawii grown in the absence of cholesterol did not. Incubation of A. laidlawii cells, grown in the absence of cholesterol, with a cholesterol-Tween 80 mixture reestablished the ability of the cells to bind cereolysin. Treatment of erythrocyte membranes and A. laidlawii cells containing cholesterol with cholesterol oxidase (EC 1.1.3.6, *Brevibacterium* sp.) abolished the ability of these membranes to bind cereolysin and inhibit the hemolytic activity of the toxin. Cereolysin could bind to and alter the permeability of both right-side-out ***ghosts*** and inside-out vesicles prepared from human erythrocytes, in agreement with other data that cholesterol is present on both sides of the erythrocyte membrane. Cereolysin caused the release of [¹⁴C]glucose from liposomes containing cholesterol, and this release was dependent on the amount of cholesterol in the liposomes.

AB The following evidence supports the concept that cholesterol in membranes is the ***receptor*** and target site for the cytolytic action of [Bacillus cereus] cereolysin. Of the various phospholipids, gangliosides and steroids tested, only . . . cereolysin and inhibit the hemolytic activity of the toxin. Cereolysin could bind to and alter the permeability of both right-side-out ***ghosts*** and inside-out vesicles prepared from human erythrocytes, in agreement with other data that cholesterol is present on both sides of. . . .

ORGN Classifier
Enterobacteriaceae 06702
Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria ; Microorganisms
Taxa Notes
Bacteria , Eubacteria, Microorganisms

ORGN Classifier
Irregular Nonsporing Gram-Positive Rods 08890
Super Taxa
Actinomycetes and Related Organisms; Eubacteria; ***Bacteria*** ;
Microorganisms
Taxa Notes
Bacteria , Eubacteria, Microorganisms

ORGN Classifier
Acholeplasmataceae 07511
Super Taxa
Mycoplasmatales; Mycoplasmas; Eubacteria; ***Bacteria*** ;
Microorganisms
Taxa Notes
Bacteria , Eubacteria, Microorganisms

L7 ANSWER 59 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
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AN 1978:140550 BIOSIS <>LOGINID::20091202>

DN PREV197865027550; BA65:27550

TI EFFECTS OF CHOLERA ENTERO TOXIN ON CATECHOLAMINE STIMULATED CHANGES IN
CATION FLUXES CELL VOLUME AND CYCLIC AMP LEVELS IN THE TURKEY ERYTHROCYTE.

AU RUDOLPH S A [Reprint author]; SCHAFER D E; GREENGARD P

CS DEP PHARMACOL, SCH MED, CASE WEST RESERVE UNIV, CLEVELAND, OHIO 44106, USA

SO Journal of Biological Chemistry, (1977) Vol. 252, No. 20, pp. 7132-7139.
CODEN: JBCHA3. ISSN: 0021-9258.

DT Article

FS BA

LA ENGLISH

AB Treatment of turkey erythrocytes with cholera enterotoxin [from *Vibrio cholerae*] caused increases in basal cyclic[cAMP levels, in the sensitivity of cAMP levels to .beta.-adrenergic stimulation and in the maximum level of cAMP attainable with catecholamines. These responses were first detected in the period from 30-60 min after addition of toxin. Na and K influxes in toxin-treated cells showed increases in basal levels and in sensitivity to catecholamines, but not in the maximal response attainable with catecholamines. Toxin-treated cells also exhibited a slow net uptake of water. In medium containing 15 mM K+, the enterotoxin caused a progressive decrease in the maximal catecholamine effect on cation fluxes; this decrease was not observed when the medium contained 2.5 mM K+. A net uptake of K+, Na+ and H₂O, similar to that which occurs in response to catecholamines or cAMP at high extracellular K+ concentrations, also occurs with cholera enterotoxin; this accumulation of water and cations apparently has some feedback effect on cation fluxes, rendering them insensitive to cAMP. Cholera enterotoxin did not appear to affect the hormone- ***receptor*** interaction as judged by the binding of [³H]alprenolol, a .beta.-adrenergic antagonist. ***Ghosts*** prepared from control and toxin-treated erythrocytes had similar capacities and affinities for this ***ligand***. On the basis of the [³H]alprenolol binding data and the effect of various concentrations of isoproterenol on cation fluxes, normal cells appear to require occupancy of about 10 receptors to activate the cAMP-dependent flux mechanism, but in toxin-treated cells, occupancy of a single ***receptor*** appears to be sufficient. Cholera enterotoxin probably causes an alteration in the ***receptor***-adenylate cyclase interaction and an increase in basal adenylate cyclase activity. The effects of cholera enterotoxin on cation fluxes and volume changes in the turkey erythrocyte appear to be accounted for by these effects on cAMP accumulation.

AB. . . has some feedback effect on cation fluxes, rendering them insensitive to cAMP. Cholera enterotoxin did not appear to affect the hormone- ***receptor*** interaction as judged by the binding of [³H]alprenolol, a .beta.-adrenergic antagonist. ***Ghosts*** prepared from control and toxin-treated erythrocytes had similar capacities and affinities for this ***ligand***. On the basis of the [³H]alprenolol binding data and the effect of various concentrations of isoproterenol on cation fluxes, normal. . . require occupancy of about 10 receptors to activate the cAMP-dependent flux mechanism, but in toxin-treated cells, occupancy of a single ***receptor*** appears to be sufficient. Cholera enterotoxin probably causes an alteration in the ***receptor***-adenylate cyclase interaction and an increase in basal adenylate cyclase activity. The effects of cholera enterotoxin on cation fluxes and volume.

. .

ganglioside, with substantial amounts of N-acetylgalactosaminyl-[N-acetylneuraminy]-galactosylglucosylceramide (GM2) and smaller amounts of other higher homologues also present. Native GM1 was not detected in any of these preparations. Examination of the relative capacities of various exogenously added radiolabeled sphingolipids to bind to the cells indicated that GM2 and glucosylsphingosine were accumulated by the cells to extents comparable to GM1. Galactosylsphingosine and sulfatide also exhibited significant, although lesser, binding affinities for the cells. The adipocytes appeared to nonspecifically bind exogenously added GM1; saturation of binding sites for GM1 could not be observed up to the highest concentration tested (2 .times. 10⁻⁴ M), wherein about 7 .times. 10⁹ molecules were associated with the cells. Essentially all exogenously added GM1 was found bound to the plasma membrane ***ghost*** fraction. Investigation of the biological responses of the cells confirmed their sensitivities to CT and epinephrine-stimulated lipolysis, as well as the lag period displayed during the toxin's action. While the toxin's lipolytic activity can be enhanced by prior treatment of the fat cells with GM1, added GM1 enhanced only the subsequent rate, but not the extent, of toxin stimulated glycerol release (lipolysis) from the cells. The ability of GM1 to enhance the toxin's activity at saturating or low toxin concentrations was unconfirmed. The limited ability of added GM1 to enhance the toxin's activity appeared in a unique bell-shaped dose-response manner. The inability of high levels of GM1 to stimulate a dose of toxin that was ineffective on native cells suggests that the earlier reported ability of crude brain gangliosides to accomplish this was due to some component other than GM1 in the crude extract. While several glycosphingolipids and some other carbohydrate-containing substances that were tested lacked the ability to mimic the enhancing effect of GM1, 4-methylumbelliferyl-beta-D-galactoside exhibited an effect similar to, although less pronounced than, that of GM1. These findings do not support the earlier hypotheses that GM1 is CT's naturally occurring membrane ***receptor*** on native fat cells, and the ability of exogenously added GM1 to enhance the toxin's lipolytic activity represents the specific creation of additional natural receptors on adipocytes. Alternative explanations are proposed which do not invoke GM1 as the native ***receptor*** for CT but which may account for the observed effects.

TI LIPOLYTIC ACTION OF CHOLERA TOXIN ON FAT CELLS REEXAMINATION OF THE CONCEPT IMPLICATING GM-1 GANGLIOSIDE AS THE NATIVE MEMBRANE

RECEPTOR .

AB. . . on isolated [rat] fat cells was examined. Analyses of the ganglioside content and composition of intact fat cells, their membranous ***ghosts*** and the total particulate fraction of these cells indicate that N-acetylneuraminylgalactosylglucosylceramide (GM3) represents the major ganglioside, with substantial amounts of. . . .times. 10⁹ molecules were associated with the cells. Essentially all exogenously added GM1 was found bound to the plasma membrane ***ghost*** fraction. Investigation of the biological responses of the cells confirmed their sensitivities to CT and epinephrine-stimulated lipolysis, as well as. . . pronounced than, that of GM1. These findings do not support the earlier hypotheses that GM1 is CT's naturally occurring membrane ***receptor*** on native fat cells, and the ability of exogenously added

GM1 to enhance the toxin's lipolytic activity represents the specific creation of additional natural receptors on adipocytes. Alternative explanations are proposed which do not invoke GM1 as the native

receptor for CT but which may account for the observed effects.

ORGN Classifier

Bacteria 05000

Super Taxa

Microorganisms

Taxa Notes

Bacteria , Eubacteria, Microorganisms

ORGN Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates,. . .

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AN 1977:125713 BIOSIS <>LOGINID::20091202>>

DN PREV197763020577; BA63:20577

TI MUTATIONAL CHANGE OF MEMBRANE ARCHITECTURE MUTANTS OF ESCHERICHIA-COLI
K-12 MISSING MAJOR PROTEINS OF THE OUTER CELL ENVELOPE MEMBRANE.

AU SCHWEIZER M; SCHWARZ H; SONNTAG I; HENNING U

SO Biochimica et Biophysica Acta, (1976) Vol. 448, No. 3, pp. 474-491.
CODEN: BBACAQ. ISSN: 0006-3002.

DT Article

FS BA

LA Unavailable

AB Mutants of *E. coli* were analyzed which lack 2 of the major proteins of the outer cell envelope membrane. The 2 proteins I and II*, normally are present at high concentrations (.apprx. 105 copies/cell). In such mutants, as compared with wild type, the phospholipid-to-protein ratio in the outer membrane increased by a factor of 2.3, causing a considerable difference in density between wild type and mutant membranes. The concentrations of 2 other major components of the outer membrane, lipopolysaccharide and Braun's lipoprotein, did not change. The protein-deficient mutants do not exhibit gross functional defects *in vitro*. An increased sensitivity to EDTA and a slightly increased sensitivity to dodecyl sulfate (but not to deoxycholate or Triton X-100) was observed, loss of periplasmic enzymes was not found, and other differences to wild type are marginal. The mutants grow with normal morphology. It is not possible to prepare ***ghosts*** (particles of size and shape of the cell without murein, surrounded by a derivative of the outer membrane, and possessing the major proteins of this membrane) from them. The proteins in question are apparently required for the shape maintenance phenomenon in ***ghosts***, and the mutants indicate that these proteins are not involved in the expression of the genetic information specifying cellular shape. Freeze-fracturing showed that in mutant cells, in sharp contrast to wild type, the predominant fracture plane is within the outer membrane. The concentration of the densely packed particles at the outer and concave leaflet of this fracture plane is greatly reduced. It was not possible to establish that 1 or the other protein is part of these particles since these ultrastructural differences were not apparent in mutants missing only 1 of the proteins. The loss of 2 major proteins and the concomitant increase of phospholipid concentration apparently changed the architecture of the outer membrane from a highly oriented structure with a large fraction of protein-protein interaction, to 1 predominantly exhibiting planar lipid bilayer characteristics. *E. coli* thus assemble different outer membranes,

indicating that outer membrane formation does not constitute a highly ordered or strictly sequential assembly-line process.

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IT Miscellaneous Descriptors

PHOSPHO LIPID LIPO POLY ***SACCHARIDE*** MUREIN PERIPLASMIC ENZYME
MORPHOLOGY GENE EXPRESSION CONCAVE LEAFLET STRUCTURE

ORGN Classifier

Bacteria 05000

Super Taxa

Microorganisms

Taxa Notes

Bacteria , Eubacteria, Microorganisms

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STN DUPLICATE 16

AN 1976:216651 BIOSIS <>LOGINID::20091202>>

DN PREV19762046651; BA62:46651

TI MULTIPLE STEPS DURING THE INTERACTION BETWEEN COLI PHAGE LAMBDA AND ITS
RECEPTOR PROTEIN IN-VITRO.

AU ROA M; SCANDELLA D

SO Virology, (1976) Vol. 72, No. 1, pp. 182-194.

CODEN: VIRLAX. ISSN: 0042-6822.

DT Article

FS BA

LA Unavailable

AB Phage .lambda. and its purified ***receptor*** protein [Escherichia coli] interact in vitro even when the phage is not inactivated; in the absence of detergent the ***receptor*** is relatively insoluble and it leads to the formation of phage aggregates. Under conditions where the phage is inactivated by the ***receptor***, only a small fraction (about 30%) of its DNA becomes sensitive to nucleases. Ejection of the DNA apparently is almost complete upon sucrose gradient centrifugation since clear separation of ***ghosts*** and DNA can be obtained. It is possible to recover from the gradients some inactive phage particles which have not yet ejected their nucleic acid. The different steps occurring in vitro, i.e., reversible interaction, phage inactivation and DNA ejection, are correlated with the 1st steps of phage infection in vivo.

TI MULTIPLE STEPS DURING THE INTERACTION BETWEEN COLI PHAGE LAMBDA AND ITS
RECEPTOR PROTEIN IN-VITRO.

AB Phage .lambda. and its purified ***receptor*** protein [Escherichia coli] interact in vitro even when the phage is not inactivated; in the absence of detergent the ***receptor*** is relatively insoluble and it leads to the formation of phage aggregates. Under conditions where the phage is inactivated by the ***receptor***, only a small fraction (about 30%) of its DNA becomes sensitive to nucleases. Ejection of the DNA apparently is almost complete upon sucrose gradient centrifugation since clear separation of ***ghosts*** and DNA can be obtained. It is possible to recover from the gradients some inactive phage particles which have not. . .

ORGN Classifier

Viruses 03000
Super Taxa
Microorganisms
Taxa Notes
Microorganisms, Viruses
ORGN Classifier ***Bacteria*** 05000
Super Taxa
Microorganisms
Taxa Notes
Bacteria , Eubacteria, Microorganisms

L7 ANSWER 64 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 1978:66865 BIOSIS <>LOGINID::20091202>>
DN PREV197815010365; BR1:10365
TI ULTRASTRUCTURAL AND BIOCHEMICAL EXAMINATIONS OF CELL WALL ***GHOSTS***
OF ROUGH AND SMOOTH STRAINS OF ESCHERICHIA-COLI.
AU LAM J; IRVIN R T; COSTERTON J W
SO Canadian Federation of Biological Societies Proceedings, (1976) Vol. 19,
pp. 12.
ISSN: 0068-869X.
DT Article
FS BR
LA Unavailable
TI ULTRASTRUCTURAL AND BIOCHEMICAL EXAMINATIONS OF CELL WALL ***GHOSTS***
OF ROUGH AND SMOOTH STRAINS OF ESCHERICHIA-COLI.
IT Miscellaneous Descriptors
ABSTRACT FREEZE ETCHING LIPO POLY ***SACCHARIDE*** OUTER MEMBRANE
ORGN Classifier ***Bacteria*** 05000
Super Taxa
Microorganisms
Taxa Notes
Bacteria , Eubacteria, Microorganisms

L7 ANSWER 65 OF 68 MEDLINE on STN
AN 1975046845 MEDLINE <>LOGINID::20091202>>
DN Published ID: 4139715
TI Use of the ***avidin*** - ***biotin*** complex for specific staining
of biological membranes in electron microscopy.
AU Heitzmann H; Richards F M
SO Proceedings of the National Academy of Sciences of the United States of
America, (1974 Sep) Vol. 71, No. 9, pp. 3537-41.
Journal code: 7505876. ISSN: 0027-8424.
Report No.: NLM-PMC433809.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197501
ED Entered STN: 10 Mar 1990
Last Updated on STN: 6 Feb 1995
Entered Medline: 25 Jan 1975
AB To expand the electron microscopist's options in localization and
visualization, a new and general staining technique has been tested. The
avidin - ***biotin*** complex serves as a coupling between the

- electron-dense marker, ferritin, and points of interest in biological samples. When specific cellular components are tagged with ***biotin***, those components may be visualized with ferritin-linked ***avidin***. Because of the remarkably strong affinity of ***avidin*** and ***biotin*** (characterized by an association constant of 10(15) M(-1)), the staining is rapid and stable. The preparation of ferritin-***avidin*** conjugate is described, and examples are presented of the application of this complex to ***biotin*** -tagged membranes. The ***ghosts*** of Acholeplasma laidlawii have been treated with biotinyl-N-hydroxysuccinimide ester to label protein amino groups. Erythrocyte membrane oligosaccharides have been oxidized by periodate or by galactose oxidase, and the resulting aldehydes labeled with ***biotin*** hydrazide. The ***avidin*** - ***biotin*** complex in electron microscopy seems especially appropriate for sequential staining procedures, as well as for visualization of reaction sites of ***biotin*** -labeled, low-molecular-weight reagents.
- TI Use of the ***avidin*** - ***biotin*** complex for specific staining of biological membranes in electron microscopy.
- AB . . . To expand the electron microscopist's options in localization and visualization, a new and general staining technique has been tested. The ***avidin*** - ***biotin*** complex serves as a coupling between the electron-dense marker, ferritin, and points of interest in biological samples. When specific cellular components are tagged with ***biotin***, those components may be visualized with ferritin-linked ***avidin***. Because of the remarkably strong affinity of ***avidin*** and ***biotin*** (characterized by an association constant of 10(15) M(-1)), the staining is rapid and stable. The preparation of ferritin-***avidin*** conjugate is described, and examples are presented of the application of this complex to ***biotin*** -tagged membranes. The ***ghosts*** of Acholeplasma laidlawii have been treated with biotinyl-N-hydroxysuccinimide ester to label protein amino groups. Erythrocyte membrane oligosaccharides have been oxidized by periodate or by galactose oxidase, and the resulting aldehydes labeled with ***biotin*** hydrazide. The ***avidin*** - ***biotin*** complex in electron microscopy seems especially appropriate for sequential staining procedures, as well as for visualization of reaction sites of ***biotin*** -labeled, low-molecular-weight reagents.
- CT Acholeplasma laidlawii: CH, chemistry
****Avidin***
*** Bacterial Proteins: AN, analysis***
Biotin
Carbohydrates: AN, analysis
Carbon Radioisotopes
Erythrocytes: CH, chemistry
Ferritins
Immunodiffusion
Ligands
*Membranes
Membranes: CH, chemistry
*Microscopy, Electron: MT, methods
*Ovalbumin
- RN ***1405-69-2 (Avidin)*** ; ***58-85-5 (Biotin)*** ; 9006-59-1 (Ovalbumin); 9007-73-2 (Ferritins)
- CN 0 (***Bacterial*** Proteins); 0 (Carbohydrates); 0 (Carbon

Radioisotopes); 0 (Ligands); 0 (Proteins)

L7 ANSWER 66 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 1974:132516 BIOSIS <>LOGINID::20091202>>
DN PREV197457032216; BA57:32216
TI CELL ENVELOPE AND SHAPE OF ESCHERICHIA-COLI STRAIN K-12 THE ***GHOST***
MEMBRANE.
AU HENNING U; HOEHN B; SONNTAG I
SO European Journal of Biochemistry, (1973) Vol. 39, No. 1, pp. 27-36.
CODEN: EJBCAI. ISSN: 0014-2956.
DT Article
FS BA
LA Unavailable
TI CELL ENVELOPE AND SHAPE OF ESCHERICHIA-COLI STRAIN K-12 THE ***GHOST***
MEMBRANE.
IT Miscellaneous Descriptors
 PHOSPHO LIPID LIPO POLY ***SACCHARIDE*** PROTEINS MUREIN
ORGN Classifier
 Microorganisms 01000
 Super Taxa
 Microorganisms
 Taxa Notes
 Microorganisms
ORGN Classifier
 Bacteria 05000
 Super Taxa
 Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

L7 ANSWER 67 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
AN 1970:98551 CAPLUS <>LOGINID::20091202>>
DN 72:98551
OREF 72:17865a,17868a
TI Specific inhibition of endotoxin coating of red cells by a human
erythrocyte membrane component
AU Springer, Georg F.; Huprikar, Shankar V.; Neter, Erwin
CS Dep. of Immunochem. Res., Evanston Hosp., Evanston, IL, USA
SO Infection and Immunity (1970), 1(1), 98-108
CODEN: INFIBR; ISSN: 0019-9567
DT Journal
LA English
AB A fraction from human erythrocyte ***ghosts*** was isolated which
prevents the attachment of unheated as well as heated lipopolysaccharides
of gram-neg. ***bacteria*** to red cells. This material has no
significant inhibitory effect either toward the Vi antigen of gram-neg.
 bacteria or towards the group and common antigens of the gram-
pos.
 bacteria investigated. It interacts with lipopolysaccharides and
not with erythrocytes, it forms complexes with and blocks those groupings
of lipopolysaccharides which attach to red cells. The effect of the
 receptor is phys. and not enzymic. The interaction of the
 receptor with the lipopolysaccharides is reversible, and the
 receptor removes lipopolysaccharides fixed to red cells. An
equil. of lipopolysaccharide distribution between cells and

receptor is established when ***receptor*** -
lipopolysaccharide complexes are incubated with red cells. The ***receptor*** is labile toward heat and toward deviation of the H+ concn. from neutrality; aldehydes destroy its inhibitory activity.

AB A fraction from human erythrocyte ***ghosts*** was isolated which prevents the attachment of unheated as well as heated lipopolysaccharides of gram-neg. ***bacteria*** to red cells. This material has no significant inhibitory effect either toward the Vi antigen of gram-neg. ***bacteria*** or towards the group and common antigens of the gram-pos.

bacteria investigated. It interacts with lipopolysaccharides and not with erythrocytes, it forms complexes with and blocks those groupings of lipopolysaccharides which attach to red cells. The effect of the ***receptor*** is phys. and not enzymic. The interaction of the ***receptor*** with the lipopolysaccharides is reversible, and the ***receptor*** removes lipopolysaccharides fixed to red cells. An equil. of lipopolysaccharide distribution between cells and ***receptor*** is established when ***receptor*** -

lipopolysaccharide complexes are incubated with red cells. The ***receptor*** is labile toward heat and toward deviation of the H+ concn. from neutrality; aldehydes destroy its inhibitory activity.

IT Toxins
RL: PROC (Process)
(erythrocyte binding of, ***receptor*** in)

IT ***Bacteria***
(lipopolysaccharides of gram-neg., erythrocyte ***receptor*** for)

IT Lipopolysaccharides
RL: PROC (Process)
(of ***bacteria***, erythrocyte binding of)

IT Erythrocytes
(toxin binding by, ***receptor*** in)

L7 ANSWER 68 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
AN 1970:108292 CAPLUS <>LOGINID::20091202>>
DN 72:108292
OREF 72:19565a,19568a
TI ***Receptor*** specificity during the interaction of Escherichia coli
bacteria with "shades" of T4 phage
AU Guseinov, R. D.
CS Inst. Obshch. Genet., Moscow, USSR
SO Doklady - Akademiya Nauk Azerbaidzhanskoi SSR (1969), 25(7), 72-6
CODEN: DAZRA7; ISSN: 0002-3078
DT Journal
LA Russian
AB The shades of T4 phages are able to kill only sensitive cells. Their action spectrum is the same as that of intact phage particles. E. coli strain B/4 resistant to T4 phage was changed to a sensitive one after 10-min treatment with EDTA at 37.degree.. Various hypotheses of reaction mech. are discussed.

TI ***Receptor*** specificity during the interaction of Escherichia coli
bacteria with "shades" of T4 phage

IT Viruses, ***bacterial***
(T 4, Escherichia coli interaction with ***ghosts*** of,
receptor specificity in)

IT Escherichia coli

(***bacteriophage*** ***ghost*** interaction with,
receptor specificity in)